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CASES ILLUSTRATING URETERAL SURGERY.

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Case I.—Miss M. L., aged twenty-six years, entered the General Memorial Hospital with a uterine fibromyoma extending as high as the umbilicus. The tumor had grown rapidly during the previous six months, causing marked pressure symptoms. Operation, June 3, 1899. Both ovaries and tubes being normal were not removed. The tumor extended between the folds of the left broad ligament, displacing the uterine artery and ureter to such a degree that both were ligated and divided together before the accident was discovered. The proximal end of the ureter was freed from the ligature and was identified by the escape of urine. It was surrounded by gauze, the entire uterus was removed, and an examination of the ureter was made with a view to immediate repair of the injury. As the ends could be easily approximated, I decided to perform uretero-ureteral anastomosis by the lateral method advocated by Van Hook and Kelly—ligating the distal end and making a slit in the duct one quarter of an inch below the point of ligation, through which the upper end was drawn by traction sutures of fine silk. Sero-serous sutures were introduced in the usual manner, and the united ends were covered with a flap of peritoneum. The peritoneal flaps were sutured and the subperitoneal space drained per vaginam. Time of operation about one hour. The convalescence was normal, with the exception of a rise in temperature on the third and nineteenth days (102° F.). The daily amount of urine varied from thirty to forty ounces until the sixth day, when a leakage was noted. This was neither constant nor considerable. The patient was kept in the hospital for six weeks, in the hope that the fistula would close. The vaginal wound healed so that the opening could not be identified. The dribbling occurred only when the patient was up and about, so that it was inferred that there was a small valve-like opening at the point of anastomosis, and not a complete separation of the ends of the ureter. Thirty ounces of urine were passed daily. I saw the patient at my

office a month later. She was in excellent health, and was not annoyed by the leakage, which was slight and did not occur at all during the night. She subsequently went abroad, and I have not seen her since, but her physician informed me a year after the operation that the fistula had entirely closed.

CASE II.—Mrs. R., aged fifty-nine years. Operation at the Memorial Hospital, June 17, 1900. Double intraligamentary dermoid cyst, with universal intestinal and intrapelvic adhesions. The uterus was so intimately attached to the growths that it was necessary to remove it. After freeing the cyst on the right side the uterine artery was ligated, the cervix divided, and the entire mass was reflected to the opposite side, in order to enucleate the growth in the left broad ligament. The left ureter was not identified until removal of the tumor, when it was found that it had been torn across. The stump of the cervix was removed and the ureter was examined. It was found that the distal end was so much dilated that the proximal end could easily be invaginated into it to the distance of an inch without danger of constriction. This was done, and the ends were secured with sero-serous sutures. It was impossible to secure a peritoneal flap, as the whole pelvis had been denuded of peritoneum. The repaired ureter now extended across the middle of the pelvic cavity without any support, so that the entire pelvis was packed with gauze, on which the ureter rested. While inserting the gauze, traction was inadvertently made upon the ureter and the ends pulled apart, so that it was necessary to do the work over again. Peritoneal flaps were secured with some difficulty, and the raw surface was shut off from the general cavity, with vaginal drainage. The patient was under ether an hour and a half and had considerable shock, but rallied quickly and made an uninterrupted recovery, passing a normal amount of urine from the outset. She was discharged four weeks after operation, and has since remained in good health.

CASE III.—Mrs. K., aged thirty-one years, was referred to me on account of a supposed cyst in the anterior vaginal wall. On examination I found a soft, doughy tumor, the size of a grape-fruit, occupying the vesico-uterine pouch, displacing the uterus backward and the bladder forward. It was easily palpated above the symphysis and extended downward almost to the meatus. The patient was quite anæmic, with a loud systolic bruit. Operation at the Memorial Hospital, March 7, 1900. An explorative incision was made into the supposed cyst, and a bloody fluid escaped. On enlarging the incision and introducing the finger an irregular cavity was felt, filled with soft, gelatinous masses, fully a handful of which were scooped out; there seemed to be no distinct capsule. The bleeding was so profuse that I was obliged to tampon the cavity and desist, as the patient became pulseless and was revived only with heroic treatment.

The microscopical diagnosis was myxo-angioma, with probably malignant degeneration (sarcoma).

Two weeks later the mass was only one-third its original size, and the patient was so much improved that I made a second attempt to remove it. A free incision was made over the growth, and it was enucleated by blunt dissection from above downward until the base of the bladder was reached, when it was dissected out with scissors. To my great chagrin, on examining the mass after removal I found attached to it a portion of the trigone, an inch square, and at least an

inch of the left ureter. I also found that the right ureter had been severed close to the bladder. Although the patient was again in collapse from loss of blood, so that rapid work was necessary, the wound in the bladder was closed with superficial and deep sutures, the end of the left ureter being turned into the bladder at a point where the tension would be least, and secured with deep and superficial sutures. The right ureter was much dilated and the tension such that I was unable to draw it down. The patient's condition was now so alarming that it seemed improbable that she would survive, so that the operation was suspended, the bed of the tumor being packed with gauze. She rallied quickly as before. A catheter was left *in situ*, and during the first week from thirty to thirty-five ounces of normal urine were discharged through it daily, with only occasional dribbling through the wound. Eventually most of the sutures gave way, and a large fistula two inches in diameter remained, involving the entire base of the bladder. I was unable to identify the end of either ureter. The patient left the hospital after six weeks, wearing a urinal.

She was in such a miserable condition that I really thought that it would have been better for her if she had not survived.

Two months later she re-entered the hospital, and I operated for repair of the fistula. It was found that the uretero-vesical anastomosis on the left side had been entirely successful. Fortunately, the end of the right ureter had become adherent at the edge of the fistula. A probe was introduced into it, and it was dissected off with the vesical mucosa, and thus turned into the bladder. The edges of the fistula were then split so that the everted mucosa could be rolled inward. The vaginal edge was denuded, the cervix uteri being utilized to fill in the upper part of the gap. Sutures were introduced in the usual manner, parallel with the long axis of the vagina, care being exercised not to include the end of the ureter. A catheter was left *in situ* for three days, and the patient subsequently passed her urine every four hours, retaining from four to six ounces. On the sixth day the nurse allowed her to go all night, so that twelve ounces accumulated in the bladder, with the inevitable result. A small fistula developed, which was easily closed a fortnight later.

The patient has since remained perfectly well, having no vesical symptoms whatever. She is a hard-working woman, and is in better health than she has been for years. When I examined her a month ago it was difficult to find the cicatrix.

Although my experience in ureteral surgery is too limited to permit any deductions with regard to operative technique, the cases reported seemed to be sufficiently instructive to justify me in briefly recording them. They at least serve to illustrate the fact that in complicated cases, especially with intraligamentary tumors, it is sometimes impossible to identify the displaced ureters before they are injured, and, therefore, that every suspicious cord, adhesion, or supposed bloodvessel should be carefully inspected before it is clamped or ligated, and again after it has been divided. When it is established beyond a doubt by the escape of urine that a ureter has been divided, nothing except the absolutely desperate condition of the patient should deter the sur-

geon from at once attempting to repair the injury, instead of resorting to the unsurgical makeshift of suturing the end of the ureter in the abdominal wound, or to the serious procedure of removing a healthy kidney from a patient already depressed by a long and bloody operation.

The well-known difficulties attending secondary operations for the cure of ureteral fistulæ, even in the hands of experts, would seem to render it imperative that an attempt should invariably be made to secure immediate repair. As regards the method to be adopted in the individual case no fixed rules can be formulated. Each must be studied separately. The technique is now sufficiently familiar, at least theoretically, but the opportunities for its application are so rare that few surgeons have an opportunity to acquire such confidence and dexterity in dealing with this complication as with others which occur in connection with abdominal surgery.

CLINICAL HISTORY OF A CASE OF BLINDNESS FROM CONGENITAL DEFORMITY OF THE OCCIPUT.[1]

By Charles A. Oliver, A.M., M.D.,
of Philadelphia.

On October 20, 1899, through the courtesy of Mr. Frank R. Harrison, of East Liverpool, Ohio, a student in the third-year class of the Department of Medicine of the University of Pennsylvania, the writer was given the opportunity to study a case of blindness in a four-year-old girl.

The patient, the fourth of five children, was born at full term after an uncomplicated labor not requiring any aid. The first child was living and well. The second was stillborn at full term, the infant being badly macerated, and reported to have been dead for at least a month previous to its birth. The third, which was also a full-term child, lived but a few minutes. The fifth was born dead. In none of these labors except the first was there any physician in attendance. The mother, who was slightly undersized in height, had never been strong. The father, not a blood relation of his wife, died from renal disease some four years before the patient was studied. His habits had been bad, he having been in the habit of drinking at least three quarts of whiskey per week for years.

No family history of hereditary disease or congenital trouble could be elicited or determined, except that an aunt on the father's side was said to have died of tumor of the breast, which, from an incomplete history given, seemed most probably to be carcinomatous in character. The mother disclaimed the existence of any disease, though it was said

[1] Read before the October, 1901, meeting of the Section of Ophthalmology of the College of Physicians of Philadelphia.

that from time to time she exhibited a number of characteristic and well-pronounced hysterical stigmata, these being associated with slight rises of temperature, during which an elevation of a single degree was sufficient to produce an attack of delirium.

The mother stated that both of her children "had since birth slept with their eyes open," and that while sleeping a bloody froth from an unknown source often escaped from their mouths. She also asserted that the patient was born with a badly shaped head, and large, prominent eyes; but that the child could see some little until it was three years of age, when it became totally blind, and the protrusion of the eyeballs grew more pronounced. She had never noticed that the patient preferred to look to one side or to the other.

The only diseases that the patient had ever had were so-called "croup" and measles, these being diagnosed and treated by a competent physician. There was not any history of epilepsy or convulsions. The child's mental condition had always been good.

Careful physical examination failed to evidence anything wrong, except that the head was disproportionately small and quite deformed posteriorly, and that the eyes were prominent, divergent, and in constant motion. The occipital protuberance was almost wanting. The occipitoparietal suture was not well closed, the thickened serrated edges of the occipital and parietal bones curving outwardly. In front of the coronal suture the frontal prominence appeared, when laterally viewed, as if slightly raised into a dome-like elevation. The superciliary ridges were quite flattened. The orbital cavities seemed somewhat shallow, especially to their nasal sides. The root of the nose was broad, and the internal commissures of the eyes were too wide apart. Both the superior and the inferior dental arches, particularly the lower one, were shallow, while the chin showed a marked recession and apparent incompleteness of development. The nasal orifices were small. The mouth, which was almost constantly kept open, was employed for breathing purposes. Both pinna, as can be in measure seen in the reproductions of the photographs, showed a number of characteristic faults.

The eyelids, which were large and freely mobile, stretched across a pair of enlarged and prominent eyeballs that enjoyed free and full movement. The right eyeball was the larger and the more prominent. The palpebral fissures were very wide and abnormally long. Both globes had their antero-posterior axes directed outwardly, the right one being the more divergent. The eyes were in a state of constant rotary nystagmic movement. They could be pushed but slightly backward. There were not any evidences, either solid, fluctuating, or pulsatile, of orbital growth or tumor.

The pupils were round, that of the left eye being three and a half millimetres in size, and that of the right one three millimetres in diameter. Repeated examinations of the irides, which were normal, showed that they were freely and equally mobile to light-stimulus, carefully thrown from every part of the ordinary visual fields; while forced attempts, by which accommodative action should be brought into play, gave most excellent and prompt iridic reactions, and strong convergence, obtained by having the subject fix in the direction of its fingertip held against its nose, produced marked pupillary contraction.

No clonic movements of either iris sphincter could be seen, though

persistently searched for. Neither pupil dilated when the skin of the back of the neck was pinched. The elevator muscles of the upper lids and orbicularis muscles were freely active. The excursions of the two eyeballs, in spite of their prominence and divergence, were good in all of the ordinary physiological directions. The external portions of the two organs failed to present any areas of analgesia or anæsthesia. Local thermometric studies proved negative.

Thorough study of the interior of the eyeballs with the ophthalmoscope failed to show a single abnormality or sign or mark of any form of inflammation. The media were clear. The chorioids, the retina, and the optic nerve heads were healthy in every detail. Their contained blood-currents were well tinted, and a venous pulse in the larger retinal stems could be readily produced by pressure upon the globes. Intraocular tension in each eye was normal.

FIG. 1.

Careful examination showed the well-known catoptric images, the movements of the reflexes proving that there was a true accommodative play in each eye. Transillumination, as far as could be usefully employed in such a young subject, did not reveal anything abnormal.

Repeated study, with every practical form of objective and subjective test that could be conveniently employed at the time, conclusively showed that the little patient could not see even the strongest light-stimulus with either eye. Her behavior and her manner were typically those of one who is blind.

The unusual pupil signs, such as the hemianopic iris inaction (or Wernicke's sign), and the Knies pupil-symptom were unsuccessfully searched for.

In spite of the extreme youth of the patient, her mental condition was so excellent that more or less perfect essays were unsuccessfully made to discover some of the higher psycho-physiological conditions,

such as mind-blindness, visual hallucinations, word-deafness, etc. There were not the slightest evidences of any hemianopsias. There was a most probable existence of a true cortical visual amnesia, the little patient being almost constantly unable to revive visual memory pictures.

Physical examination showed that the various organs were healthy. The thyroid glands were not enlarged. There were not any cardiac or pulmonic complications. Both the deep and the superficial reflexes, as far as could be practically obtained, seemed normal for age and condition. The teeth, which were the primary ones, were irregularly placed and badly cared for: they did not exhibit any coarse signs of general hereditary or acquired disease. Examination of the nasal cavities revealed, as was expected from the constant mouth-breathing, the presence of adenoids; these sprang from the lateral walls of the anterior ethmoid cells, and almost completely filled the vault of the nose. Examination of the urine was negative.

FIG. 2.

Although at this single visit almost every ocular symptom that was deemed of the least importance for the determination of the cause of the blindness was searched for, yet the child was admitted into the wards of Wills Eye Hospital in order that the writer might at his leisure restudy this now, to him, most interesting case, thus obtaining in its entirety the above report of the conditions.

Some careful cephalic measurements made at this time were mislaid, though, fortunately, two photographs (got by much diplomacy and with much trouble by Mr. Harrison), shown in the accompanying reproductions, illustrate the general facial appearances very well,[1] and thus, with the deterioration, offer themselves as no mean substitutes.

[1] It will be noticed that the hair has been intentionally dressed in such a way as to conceal the posterior portion of the head.

During the child's two or three days' residence in the hospital Dr. W. W. Bulette, of Pueblo, Col., who happened to be visiting the writer at the time, kindly removed all of the adenoid tissue.

Tapping of the spinal fluid and cranial and spinal trephining were thought of, but no opportunity was given to put these procedures into practice.

Several months later the child was said to have died from an intercurrent and unrelated disease. Autopsy, though strenuously sought for, could not be obtained. The other child was not studied.

REMARKS. Unlike most of the cases of blindness found in association with deformed crania from improper synostosis, with its deficiencies and overdevelopments, this extremely rare occipital type of osseous disease failed to exhibit many coarse motor changes in and around the eye.

The family history of a dissipated father, whose ancestral tree was bad, and a probably infected mother gave answer in great measure for the obtainment of such a product. In view of Friedenwald's observation of the preponderance of the male subject in similar cases, the sex of the case—a female—is of interest.

The presence of the adenoids cannot in any way account for such a grouping of symptoms.

In this character of subject the early want of proper binocular stimulus from imperfect visual perception, with a constantly increasing interference of sensory receipt, soon disturbs much of the interrelation of the two motor portions of the apparatus that are intended for interassociation of vision, causing the eyeballs to diverge and nystagmus to appear.[1]

Far different is this from that which is seen in the oxycephalic or even the gross hypsicephalic types of cranial deformation, with their characteristic steeple-shaped or dome-like heads. In this class of degenerates the signs of disturbance in the visual apparatus are marked by gross inflammatory and degenerative changes taking place in the orbital contents. In this type, in which there is an improper union of the parietal with the occipital and temporal bones, with compensatory osseous overdevelopment along the sagittal suture and in the position of the anterior fontanelle (giving the head the appearance of a sugar-loaf), the primary changes upon delicate structures, such as nerve, vessel, lymph-channel, and even the eyeball itself, soon set into activity a whole chain of low-grade inflammatory and pressure degenerations which result in the functional destruction of the ocular globes and optic nerves. Externally, grossly proptosed, widely divergent, coarsely degenerate in many of its parts, and but indifferently mobile, the worst

[1] It is possible that this last symptom is also due to an imperfect development of the paths between the visual cortex and the so-called primary optic ganglia.

cases of this type of ocular involvement to the veriest examples, with their signs of slight prominence, fixed irides to light-stimulation, and optic nerve degeneration, may be seen.

The scaphocephalic or boat-shaped malformation of the skull, with its extremely broad forehead, caused by an improper union of the sagittal suture between the medial margins of the parietal bones, is more disposed to give rise to all of the well-known symptoms of optic neuritis, followed by atrophy. Prominent, sightless, and divergent eyes, with more or less fixed irides to light-stimulation, in subjects of fair intelligence at best—victims of convulsive seizures, as a rule—characterize the most pronounced cases in this class of abnormality.

In the leptocephalic types, in which the heads are extremely small, caused by a premature union of the fronto-sphenoidal suture between the alæ of the frontal and sphenoid bones, post-neuritic atrophy is extremely apt to appear very early in life.

The most curious trigonocephalic or three-cornered type of cranial deformity, with its small end situated anteriorly, which is due to an improper union of the frontal and parietal bones or the frontal bones at the coronal or frontal suture, has post-neuritic atrophy for its most pronounced ocular sign.

In the present case—an extremely rare example of what might with propriety be termed the occipital or occipitoparietal type of cranial malformation, in which the configuration of the posterior portion of the head is that of a flattened, but slightly curved surface, extending irregularly in an upward, forward plane to meet the frontal protuberance—the most marked ocular signs are almost wholly sensory in character. Vision in each eye is nearly or entirely lost. The orbits are shallow, particularly at their postero-mesial parts. The eyeballs are but slightly proptosed, somewhat enlarged, and enjoy full freedom of movement. The entire motor apparatus of the exterior of the eyes, with the exception of a few minor discrepancies of probable improper nuclear action, is in proper working order. The pupils are but slightly if any oversized. The irides are prompt to light-stimulus, efforts for accommodation, and convergence. The ciliary muscles are active. The eye-grounds, in every detail of neuronic, vascular, and lymph structure, appear normal; in fact, the eyeballs, with their entire adnexa, are healthy, and perform their functioning duties properly.[1]

This complexus of symptoms, with its absolute blindness[2] and con-

[1] Careful comparison of Stood's well-known though imperfectly reported case in a thirty-three-year-old man, shows that his patient was subject to convulsive attacks which might have developed in the writer's case had it lived longer.

[2] As may be inferred throughout this paper, the writer is disposed to concur with Friedenwald's suggestion that "in many cases the optic nerve affection is due to temporary increase in intracranial pressure." In this type the query arises, May not the probable cortical and

comitants of slight globular protrusion, divergence, and rotary nystagmus as the only ocular signs, constitute a most remarkable clinical picture. In it is seen a blindness the proving of which necessitates a most careful study of every possible direct and indirect ocular detail; a blindness that from the ocular signs and associated conditions may be safely assumed as intracranial in type, and, most probably, until autopsy proves to the contrary, cortical in character. In such cases it is fairly certain that there is a healthy receiving material which is properly functioning: all of the ethereal wave vibrations that ordinarily give rise to the perception of color being duly received and transmitted to an intracranial position that is intended for use in ultimate perception. This lower cerebral centre—known as the visual cortex—is, unfortunately in such cases, as in the one herein detailed, of such imperfect development and of so feeble a functioning and resisting power that it early loses much of its physiological activity, and, sooner or later, degenerates into a functionless and, at times, a useless organ.

DOUBLE HYDROCELE IN AN INFANT—PROLAPSUS OF THE RECTUM—A CASE OF OSTEOMYELITIS OF THE TIBIA—NECROSIS OF THE LOWER JAW FOLLOWING MEASLES—A CASE OF KNOCK-KNEE—CELLULITIS OF THE PENIS AND SCROTUM.[1]

By Henry R. Wharton, M.D.,
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Double Hydrocele in an Infant.

The first case I show to-day is a male infant seven weeks old, whose parents bring him to the hospital on account of an enlargement of the scrotum. They say that the swelling has developed gradually and does not increase materially when the child cries.

The most common conditions which would cause enlargement of the scrotum in an infant would be either hernia, hydrocele, or a growth such as sarcoma or tuberculosis of the testicle. Upon examination of the swollen scrotum I find that it presents an elastic feel, that I can feel the testes, which are normal in size, and that the tumor cannot be

connected neuronic inflammation and degeneration have been dependent in measure upon localized stretching and inflammatory processes taking place in and around a series of imperfectly made and quite readily disturbed tissues in the occipital region?

[1] A clinical lecture delivered at the Children's Hospital, October 16, 1901.

reduced by taxis, and that it does not increase in size when the child cries. The latter conditions will separate it from hernia; its elasticity and the presence of testes of normal size will differentiate it from sarcoma or from tuberculosis of the testes. Finally, I examine it by transmitted light, and find that it is translucent, and that I can see the testes occupying their normal position at the lower and back portion of the scrotum. The case is therefore one of double hydrocele.

This condition, which consists of a collection of serum in the tunica vaginalis testis, is not an uncommon one in children, and is chiefly of interest as regards the question of diagnosis from hernia or tumor of the testes.

As regards the treatment, I will first order for this infant a lotion of ammonium muriate, ten grains to one ounce of water, to be applied constantly to the scrotum on lint, and under its use the fluid may disappear in a week or two. If, however, it does not disappear, I shall then tap each distended tunica vaginalis with the trocar and canula and remove the fluid, repeating the tapping if they become again filled up. This usually brings about a cure of the disease, but if this should fail after tapping, I will inject a few drops of tincture of iodine, carbolic acid, or alcohol. Radical operations, such as dissecting out a portion of the sac, are rarely required in the treatment of hydrocele in infants or children.

Prolapsus of the Rectum.

This boy, seven years of age, was admitted to the hospital with the history that the rectum for some time had been coming down at stool and also when he passed his urine.

The patient has suffered from frequent urination, and an examination of the urine shows a large number of leucocytes. When I examined the patient yesterday the rectum was prolapsed, but now, when he is under the influence of an anesthetic, it does not appear.

Prolapsus of the rectum is one of the most common rectal affections of childhood, and we recognize it in several varieties. The most common and simplest form is that in which there is relaxation of the submucous connective tissue of the rectum, which allows a ring of mucous membrane to protrude from the anus upon straining at stool. In another form we have both protrusion of the mucous membrane and the other coats of the rectum. In a third variety, which fortunately is rare, there is protrusion of the mucous membrane and the other coats of the bowel, with an invagination of the upper portion of the rectum into the protrusion.

The frequency of this affection in children is accounted for by Jacobi by the presence of angular flexures in the lower portion of the large intestine, which necessitate marked straining in the passage of feces. Improper diet, which produces frequent stools, is often the cause of this

affection. This we have seen demonstrated in this hospital, patients often being sent in for operation suffering from prolapsus, in whom, while taking the regulated diet of the hospital, the protrusion fails to appear, and they recover without operation. The habit of allowing children to spend a long time upon the chamber utensil, with marked straining, also conduces to this disease. Difficulty in urination, due to the presence of a stone in the bladder or a narrow preputial orifice, giving rise to straining efforts in micturition, may also cause prolapsus of the rectum.

Prolapsus of the rectum is only likely to be confused with hemorrhoids or polypus of the rectum. The former condition, hemorrhoids, is extremely rare in childhood. Polypus of the rectum is also a comparatively rare affection. The diagnosis can usually be made without difficulty by a careful examination of the parts. In hemorrhoids there are one or more isolated tumors; in polypus the protruded tumor has a well-marked pedicle; while in prolapsus there is protruded a circular mass covered with mucous membrane with a depression in the centre. The treatment of prolapsus of the rectum depends upon its variety; in mild cases regulation of the diet and the daily injection after stools of a few ounces of a decoction of oak bark will be followed by a cure. In other cases if the skin of the buttock is drawn to one side during the act of defecation the prolapsus will be prevented from appearing.

The operative treatment which I have usually found effective in mild and persistent cases is that recommended by Allingham, which consists in the cauterization of the protruded mass with nitric acid. I will employ this procedure in this case, but before doing so, as the patient has suffered from vesical irritation, I will introduce a sound into the bladder to ascertain whether there is present a stone. If the latter were discovered, its removal by operative procedure would cure the affection without resort to operation upon the prolapsus itself. In this case I find no evidence of stone in the bladder, although I feel that the walls of the bladder are roughened, as the result of the cystitis from which the patient has been suffering. You notice that the bowel is not prolapsed at the present time, but when I stretch the sphincter it appears. I first wipe off the surface with a weak solution of green soap to free it from mucus, and then thoroughly dry it with a pad of sterilized gauze. I next apply some cosmoline around the skin of the anus, to prevent it from being cauterized if any of the acid comes in contact with it, and then with a swab dipped in nitric acid I carefully paint over the whole exposed surface, and finally cover it with sweet oil, and reduce the mass, and apply a pad of gauze over the anus and secure it in position with broad strips of adhesive plaster and a T-bandage.

The after-treatment consists in keeping the bowels quiet for twenty-four hours, and if they do not move spontaneously at this time a mild laxative

may be given. There may be no further protrusion of the rectum, or it may protrude at the first or second passage, and then finally fail to come down when the bowels are moved.

As I stated before, I have seen excellent results following this method of treatment, but if in any case a cure does not result from this procedure the actual cautery may be employed in the same manner that it is employed for the cure of the affection in adults.

A Case of Acute Osteomyelitis of the Tibia.

I am glad to have the opportunity of showing you a case of acute infectious osteomyelitis which was operated upon about ten days ago, and I bring the patient before you to-day for dressing, that I may show you the progress of the case at this period.

The patient, a boy, eleven years of age, received a fall from a fence about a week before his admission to the hospital, striking upon the left leg. He complained for a few days of pain in the left leg a short distance above the ankle. The leg became swollen and painful, and his family physician asked me to see the patient with him. When I saw him a week after the accident I found that he had a high temperature and a rapid pulse, and complained of great tenderness and pain in the left leg. The leg was swollen and painful from the ankle to the middle of the tibia. There was a slight red flush over the anterior surface of the limb, and on pressure I could detect deep fluctuation. I advised his removal to the hospital and immediate operation, as I considered it a case of acute infectious osteomyelitis.

Upon his admission to the hospital the patient was etherized, the skin of the leg was sterilized, and an incision four inches in length was made over the tibia through the inflamed tissues. As soon as the periosteum, which was entirely separated from the surface of the tibia, was incised a considerable quantity of pus escaped. The exposed surface of the tibia was white and presented the appearance of necrosed bone. The anterior surface of the tibia for three inches or more was cut away with the gouge, exposing the medullary cavity, which was found filled with pus and granulation tissue. The opening in the bone was then extended downward almost to the lower epiphysis of the tibia. The exposed cavity was next thoroughly cleared of pus and granulation tissue with the curette and with gauze pads; the cavity was then loosely packed with strips of iodoform gauze, and a sterilized gauze dressing was applied to the limb, which was held in position by a bandage.

The after-treatment of the case consisted in removing the dressings and packing on the second day and washing out the cavity thoroughly with sterilized water or weak bichloride solution, and re-applying the dressings. This was done at intervals of two days, and you see at the present time, ten days after the operation, that the wound in the bone

is covered with healthy granulations and is healing rapidly. At the same time the patient's constitutional condition is excellent, his temperature and pulse are normal, and convalescence seems to be perfectly established.

Acute infectious osteomyelitis is not an uncommon affection in children, and may follow a traumatism or exposure to cold, or may develop during the acute febrile diseases of childhood. It most frequently attacks the long bones, and the portion of the bone near the epiphysis seems especially liable to be the seat of the affection. The infection is usually a mixed one, resulting from pyogenic and specific organisms. The organisms reach the medullary cavity by way of the circulation. A traumatism or a local congestion may determine the site of the infection.

The diagnosis of the affection is not difficult if one bears in mind the possibility of this condition. From the fact that it often involves the medulla of the long bones near their epiphyses, it is sometimes confused with acute rheumatism. Acute rheumatism is rarely monarticular, and if the case be examined carefully it will be found that in osteomyelitis the greatest swelling and tenderness is not in the joint, as in rheumatism, but close to it.

If prompt treatment is not applied in cases of acute infectious osteomyelitis, death may result from septicæmia or pyæmia, and if the patient survives the acute symptoms, extensive necrosis of the affected bone results. By prompt operative treatment, such as was employed in this case, the patient is saved the risks of septic infection, and recovery takes place without the occurrence of necrosis of the bone.

Necrosis of the Lower Jaw Following Measles.

The next patient I show represents a form of acute osteomyelitis following measles, which has resulted in necrosis of a considerable portion of the lower jaw. This condition was described by the older writers as exanthematous necrosis.

The child, aged eighteen months, had measles several months ago, after which it was noticed that there was an offensive discharge from the mouth. When admitted to the hospital, a few days ago, on examination it was found that there was necrosis of a considerable portion of the lower jaw, with extensive ulceration of the surrounding gum.

This condition is not infrequently seen in poorly nourished children as a sequel of measles. In some cases, in addition to the loosening of the teeth and the necrosis of the bone, there is extensive destruction of the gum and of the adjacent soft parts. Gangrene of the cheeks or of the chin often results, giving rise to the condition which is known as noma. The gangrenous process may at the same time involve the genitals. The lesions of the soft parts and of the bone in these cases result from pyogenic and specific organisms giving rise to a mixed infection. Many

different organisms have been isolated, but those of the pyogenic and leptothrix variety have been most constantly observed. The disease not only results from measles, but may occur after scarlet or typhoid fever. Many patients die within a few days after the disease attacks the gums and jaw, the fatal termination being due to septicæmia or pneumonia. In other cases recovery may take place with destruction of the gums, the jaws, or the cheeks, producing great deformity.

The treatment which we employ in these cases is carried out in the following manner: The patient is etherized, the mouth is exposed as widely as possible by the use of a gag, the inflamed tissues are wiped free from discharge with a pad of gauze, loose teeth are removed, and any exposed necrosed bone which is loose is removed with an elevator or by means of bone forceps. Gangrenous soft parts are removed with scissors or a curette, and the raw or ulcerated surface is then cauterized with nitric acid or with the actual cautery. The patient then has the mouth thoroughly washed out at frequent intervals with a 1 to 2000 permanganate of potash solution, alternating with a solution of chlorate of potash. Stimulants and tonics are given at the same time, and a nutritious and easily assimilated diet should also be prescribed. Under this treatment a certain number of cases recover, and perforation of the cheek or chin may be prevented. In spite of treatment, however, the mortality following this affection is very high.

A Case of Knock-knee.

This colored child, four years of age, presents a marked rhachitic deformity known as knock-knee. You notice that when the internal condyles are in contact the legs are separated at the ankle for a distance of about 12 to 14 inches. You see at the same time that there is a decided flat foot. This condition may be treated by the application of braces or by an operative procedure known as osteotomy. The latter method in a patient in this class of life is the better procedure. I will in this case do an osteotomy just above the condyles of the femur—Macewen's operation.

The patient being etherized and the limbs having previously been sterilized, I make a puncture with a bistoury down to the bone about 1½ inches above the inner condyle of the femur, and pass an osteotome into this incision down to the bone, and turn it so that the blade is transverse to the long axis of bone. With the strokes of a mallet I next carefully divide the bone, keeping my incision well away from the posterior surface of the femur, to avoid possible injury of the important bloodvessels in that region. When I feel by the resistance offered to the osteotome that the bone is nearly divided, I remove the instrument, and grasping the thigh and the leg I complete the division by fracturing the remaining fibres of the bone. The limb is then brought into a

position of overcorrection, the small wound is closed by one or more sutures, and a compress of gauze saturated with the tincture of benzoin and collodion is placed over it. A layer of cotton is next applied to the ankle and the bony prominences about the knee, and a flannel bandage is applied to hold this in position. While the limb is held in a position of overcorrection a plaster-of-Paris bandage is carefully applied from the foot to the upper portion of the thigh. The same operative procedure is applied to the other limb, and a correction of the deformity is secured in the same manner.

There is usually no reaction following this operation and the plaster-of-Paris bandages are allowed to remain in position for four weeks. They are then removed and fresh bandages applied for three or four weeks longer. At the end of eight weeks union is usually quite firm, and the patient is then allowed to use the limbs in walking, without any support being applied to the limb.

Cellulitis of the Penis and Scrotum.

This infant, aged seven weeks, presents a swollen condition of the penis and scrotum, with, at the same time, numerous superficial points of ulceration over the inflamed surface. The condition is one which we recognize as cellulitis, and has existed for a week, and, according to the statement of the mother, appeared a few days after a forcible retraction of the prepuce to relieve a condition of phimosis. It is possible that this condition resulted from an infection of the tissues as the result of this procedure. I have seen retraction of the prepuce in an infant result in infection and gangrene of the prepuce, and therefore do not consider the procedure an entirely safe one unless great care is taken to thoroughly sterilize the parts and the fingers or instruments used before the procedure is employed.

In this case the condition seems at the present time to require no treatment other than great care in keeping the parts thoroughly cleansed by the use of castile soap and water and a lotion of boric acid, followed by a protective dressing of oxide of zinc ointment.

IMPACTED CALCULUS IN THE URETHRA IN CHILDREN.

A REPORT OF TWO CASES.[1]

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Vesical calculus in children, although infrequent in this country, is not of such rarity as to occasion comment when encountered. The

[1] Read before the Philadelphia Pediatric Society, November 12, 1901.

accident of impaction in the urethra is a sequel of more unusual occurrence, and having met with two such cases within a few months at the Children's Hospital has led me to make this report of them, with some remarks upon the diagnosis and treatment.

CASE I.—Edward N., white, aged three years; admitted February 13, 1901. The family and previous history were negative. For two weeks the child had been observed to strain while urinating and at stool. He had passed no urine for twenty-eight hours before admission, and had been very restless and cried a great deal during the preceding night. This morning it was noticed by the family that the abdomen, scrotum, and thighs were swollen and painful. On admission the temperature was 97.8° F., pulse 132, respirations 32. General condition fairly good. There was observed a very great enlargement of the scrotum, the swelling also involving the perineum and extending upward over the abdominal wall as high as the umbilicus, and laterally and below to about one-half inch below Poupart's ligament on either side. The prepuce and sheath of the penis were also involved in the œdema. The prepuce could not be retracted sufficiently to expose the meatus of the urethra. During the short time the child was under observation before operation, about one hour, the œdema could be seen to extend downward on the thighs in a line parallel to Poupart's ligament. A diagnosis of urinary extravasation was readily indicated by the history and the distribution of the swelling, and Dr. Wharton, who saw the case with me, expressed the opinion that the probable cause of urethral rupture was an impacted calculus. The child was speedily prepared for operation. Under ether the prepuce was split up on its dorsum and a sound passed into the urethra. A calculus was immediately met with in the spongy urethra, about two inches from the meatus. Owing to the smallness of the parts, no instrument could be introduced into the urethra that would grasp it, although a staff could be passed beside it into the bladder, evacuating a moderate amount of urine. With the child in the lithotomy position the urethra was opened upon the staff in the median line behind the scrotum, and after some little difficulty the stone seized where it lay in the bulb and extracted. Incisions were made on either side of the scrotum, and in both groins, to evacuate the extravasated urine, an English catheter was tied in the bladder through the perineum, and the wounds dressed. The stone was small, red, irregularly oval in shape, with a rough, mammillated surface—evidently a uric-acid calculus.

The patient did very well until the third day. The œdema rapidly subsided, and there was apparently no sloughing of the subcutaneous tissues as a result of the extravasation. On the morning of the 15th, after the temperature had dropped to normal, the child vomited, and in the evening the temperature rose to 106° F., falling again, after sponging, to 99° F. by the following morning, when a bright red rash developed on the body and limbs. The temperature rose again in the evening to 102.2° F., the pulse meanwhile ranging from 144 to 196, and the respirations from 32 to 50. During the two days following he vomited occasionally, and the temperature was irregular, varying from 98.2° F. to 101.4° F. The rash was strongly suggestive of scarlet fever, especially when considered in combination with the vomiting and rapid pulse. Dr. Griffith, who saw the case with Dr. Ashhurst and

myself, regarded it as probably one of scarlet fever, and the child was isolated. The local condition meanwhile progressed satisfactorily. The first attempts to dispense with the catheter on the third day were unsuccessful, and it had to be re-introduced. It was removed a couple of days later, and urine passed voluntarily through the perineal wound, and later through the penile urethra also. The temperature remained down from the 18th to the 20th, and the child seemed to be improving; but on the 20th it again rose to 105.4° F., and vomiting began again. On the 26th diffuse desquamation was noticed over the body and thighs. The child became much weaker, delirious, and very restless; the circulation failed rapidly, collapse developed, and he died the same evening.

The nature of the fatal complication was a matter of some doubt. In many ways it resembled scarlet fever, but it could not be positively stated that it was not a septic condition from urinary absorption, the rash being one of the so-called "surgical rashes," such as we occasionally see in children, and which in a children's hospital is a factor, sometimes causing much anxiety and alarm. The fact that powdered iodoform was sprinkled upon the wounds at the time of operation was also considered. The onset on the third day after admission precluded any question of intramural contagion, nor were there any other cases of scarlet fever at that time in the hospital.

Case II.—Francis D., white, male, aged three and a half years, admitted May 27, 1901. Family and previous history negative. On the evening of the 26th the child complained of pain over the bladder. This was about 5 P.M. No urine was passed from this time until admission in the afternoon of the 27th, when the house surgeon, Dr. Walker, succeeded in introducing a catheter into the distended bladder and emptying it, and on withdrawing the instrument a stone was felt in the urethra. On the following morning I examined the boy with Dr. Ashhurst, and found a calculus in the bulbous portion of the urethra. As it could not be dislodged per urethram, for the reasons mentioned in the previous case, median urethrotomy was at once performed. The stone, owing to its small size and smooth, rounded surface, was not easily extracted, but was finally removed by means of a small, curved stone-forceps. It was a smooth, oval, uric-acid calculus, measuring three-eighths by one-quarter inch, and weighed, when dried, four and two-thirds grains. An English catheter was tied in the bladder through the perineum. The temperature never rose above 100.4° F., and the child's condition remained satisfactory. The first attempts to remove the catheter from the perineum three days after operation failed, as it had to be re-introduced the next day. It was finally removed on June 3d and a silver catheter passed per urethram. The perineal wound was healed by June 9th and the child discharged shortly thereafter.

In the first case there were symptoms of stone noted before impaction, or at least obstruction, developed, viz., straining during urination and defecation, but they were misunderstood by the parents and attributed to constipation. In the second case no symptoms had been observed

before the stone engaged. We are often led to suspect the presence of stone in children from the presence of bladder irritability, when none exists, and will sound many children for stone where none is present to one where a calculus will be detected. Reflex irritability of the bladder is such a common symptom in children, as a result of phimosis, hyperacidity of the urine, rugosity of the bladder wall, etc., that the presence of a calculus cuts a comparatively small figure; but it is one the possibility of which should never be forgotten if we would save ourselves on some occasion from a mortifying oversight.

Retention in children I believe to be usually reflex in character, and not very uncommon as a result of phimosis, etc. Rupture of the urethra from traumatism, and perineal abscess the result of inflammation or injury, can cause real obstruction, as may also stricture of the urethra, which has been shown to manifest itself even in very young children as a result of gonorrhœal urethritis. Abbe[1] has reported a case of impermeable stricture of the deep urethra in a male child, aged two and a half years, developing six months after gonorrhœal urethritis, associated with two other strictures of the anterior urethra, and demanding suprapubic aspiration of the bladder and external urethrotomy. But the most frequent cause of urethral obstruction in children is probably calculus. Holmes, in his *Surgical Treatment of the Diseases of Infancy and Childhood*, published many years ago, says that when summoned to a case of retention in a child we can almost always assume it to be due to impacted calculus. It may, when overlooked, easily lead to perineal abscess as well as to extravasation. Even the passage of the catheter may not detect its presence.

The removal of an impacted calculus in children is, from the small size of the urethra, more difficult than in adults. If situated near the meatus, or anywhere in the penile urethra, it can sometimes be seized with fine forceps after a preliminary meatotomy and removed. In the deep urethra attempts may be made to push it backward into the bladder, where it can be treated by litholapaxy. Failing these attempts, the urethra must be promptly opened. When the urethra is already ruptured no time should be lost, and urethrotomy and drainage of the infiltrated tissues at once proceeded with. Judging from our experience, it will usually be found necessary to leave an English catheter in the perineal opening for several days, as some swelling of the parts is to be expected, and the presence of a raw surface over which the urine must flow also probably excites a reflex spasm of the sphincter.

[1] New York Medical Journal, 1890, vol. lii. p. 666.

A CASE OF CHRONIC LYMPHATIC LEUKÆMIA IN AN INFANT.

WITH SOME OBSERVATIONS ON THE BLOOD CONDITION IN OTHER CASES OF SPLENIC TUMOR.

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Our knowledge of the blood condition in many of the diseases of infants and children is at present hazy. Each individual case of anæmia in children carefully observed and studied may add something to our knowledge. It is with this hope that the present case is reported.

Peter M., an Italian boy, aged nine months, first came under the observation of Dr. Henry M. Fisher on May 20, 1900. The child's parents are both living and healthy; they present no indications of disease, though living in poor and squalid surroundings in the southern part of Philadelphia. There are three girls in the family, aged, respectively, twelve, ten, and three years. The eldest, who lives in Italy, is said to have had an enlarged spleen, with anæmia, from early infancy. She is still a delicate child. The other two children are healthy.

The parents said the boy presented no striking peculiarity until he was one month old, when it was noticed that he was unnaturally pale. Since that time he suffered frequently from recurring attacks of intestinal catarrh, with green stools containing much mucus. His mother was and still is nursing him at seventeen months, though her milk is scanty and is reinforced by other baby foods. Head was symmetrical; fontanelles almost closed; teeth were cut with normal rapidity. No evidence of rickets. At the first visit the spleen was enlarged extending fully four inches below the costal margin. There was no marked enlargement of either the liver or the external lymphatic glands. The heart and lungs were normal. The urine was non-albuminous and contained urates in excess. Blood smears were prepared and first examined on May 29th, though no blood count was made at that time. The slide showed a marked poikilocytosis, with many microcytes and macrocytes and a decided leucocytosis in which the lymphocytes were proportionately high. There was a considerable number of nucleated reds (normoblasts). There were no malaria parasites. The blood was subsequently examined, counted on June 15th, 25th, July 6th, November 23d, January 7, 1901, and May 23d. The results of these examinations will be found in the table on opposite page.

The child was placed upon quinine by suppository, and was also given one-half drop of Fowler's solution three times a day. During the latter part of May there was considerable diarrhœa, with green stools containing mucus. During July there was a slight improvement in the patient's condition, which seemed to follow the use of oil inunctions and eudoxine, 2 grains given every two hours. The child was advised to be given as much as possible the benefit of fresh air, and was at times taken to the sanitarium at Red Bank. The spleen had enlarged during this interval, and could now be felt to within one finger's breadth of the iliac crest, and posteriorly two fingers' breadth below

the costal margin. He was not seen again until November 21st, when the child had frequent stools, with considerable bronchial catarrh. At that time he was put upon ½ a grain of glycerin-phosphate of soda in solution with dilute phosphoric acid, ½ minim of Fowler's solution and 2 grains of protonuclein, t. d., which treatment was continued through December.

BLOOD TABLE.

	1900.					1901.	
	May 24.	June 15.	June 27.	July 6.	Nov. 23.	Jan. 7.	May 23.
Erythrocytes,	.....	3,120,000	3,080,000	Not count'd	3,360,000	.....	3,097,000
Leucocytes,	.....	105,000	54,000	" "	18,600	36,700	80,733
Hæmoglobin,	.....	20 per ct.	22 per ct.	.....	20 per ct.	30 per ct.	15 per ct.
Polynuclear,	14 per ct.	27.5 "	30 "	28 per ct.	25 "	47 "	15 "
Small lymphocytes	65 "	27 "	17 "	44 "	44 "	23.4 "	66.9 "
Large lymphocytes	18 "	30 "	44 "	18.4 "	9.6 "	20.6 "	13.8 "
Eosinophiles,	3 "	7.5 "	2 "	4.8 "	8.6 "	5.6 "	2.2 "
Myelocytes,	0 "	8 "	7 "	4 "	2.7 "	3.4 "	2.1 "
Normoblasts,	33	92	39	46	Many	35	125
Megaloblasts,	8	2	.....	5	.....	18	8
Microblasts,	3	0	.....	1	.....	3	2
Polychromatophilia	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number counted,	200	200	225	250	185	500	1000

On November 30th Dr. Charles A. Oliver examined the eye-grounds and reported " a neuroretinitis, with engorgement of both the retinal arteries and veins, with extremely pallid blood; there were no hemorrhages." When last seen, May 23d, the child had suffered a relapse, for it had improved considerably during the early winter months. It presented a peculiar clayish pallor, the lips colorless, with but little emaciation, the abdomen enlarged and protuberant, the spleen hard and very much enlarged, with no enlargement of the superficial lymphatic glands, and the edge of the liver just palpable. Since the beginning of the illness he has never had any nose-bleed. Once, in January, he coughed up a small amount of blood. There have never been intestinal or gastric hemorrhages. The child died about ten days (June 2d) after the last blood examination, from asthenia; no autopsy could be secured, as Dr. Fisher was unfortunately not in Philadelphia at the time.

A brief résumé of the appearance of the blood slides is, I think, necessary at this time. The first film prepared by Dr. Fisher and sent to me for diagnosis showed many changes in the size and form of the red, with much loss of hæmaglobin and decided leucocytosis, the percentage of lymphocytes being comparatively high, some few myelocytes present, and nucleated reds in plenty, some undergoing mitotic change. During June there was a great increase in the number of leucocytes, reaching over 100,000 per cubic mm. There were again numbers of microcytes with very pale macrocytes, with large numbers of both young and old normoblasts, many of them mitotic. There were many polychromatophilic cells; degenerative changes in the reds were common, especially in the old normoblasts. At this time the proportion of large lymphocytes was rather high. In July there was little change to note in the blood except the reduction of the total number of the leucocytes, which continued to fall until, November 23d, they were but 18,600, with a proportionate increase in small lymphocytes. In January the

leucocytes again began to rise, the count at this time being 37,600, and when last seen in May, after a lapse of some four months, during which time treatment had ceased through neglect of the parents, the white count reached 80,000. During this period of almost a year the erythrocyte count has remained almost stationary (about 3,000,000), while the hæmaglobin has been low. The last differential counts are of considerable interest. They show a distinct increase in the lymphatic elements of the blood; in fact, the last films made, as can be seen by consulting the table, showed over 80 per cent. of lymphocytes. Over 66 per cent. of these are small, while but 13 per cent. are large lymphocytes; many of them, however, resemble nucleated reds, the nucleus remaining rather reticulated. Some of the red corpuscles show a basophilic granulation; some of the lymphocytes are undergoing segmentation, and three or four were observed with bipolar nuclei. Myelocytes are present but in rather scanty numbers; almost 50 per cent. of the eosinophiles are myelocytic.

Diagnosis. In children enlargement of the spleen is common; the usual causes may be briefly stated as malaria, typhoid fever, congenital syphilis, rickets, v. Jaksch's disease, splenic anæmia, leukæmia, and acute and chronic infections. A differential diagnosis between two of these diseases at times becomes extremely difficult unless we carefully bear in mind the characteristic reaction of the infant and the child's blood in various diseases—*i. e.*, the ready response of the leucocytes, and especially the lymphocytes, to any call made upon them.

Thus *malaria* in the infant or child is usually associated with definite clinical symptoms, enlargement of the spleen, the absence of leucocytosis, the presence of the specific parasite. In chronic paludism this picture may be somewhat changed. The degree of anæmia may be severe, and corresponding changes may occur in the red cells, but there is no marked alteration in the leucocytes except a tendency to a lymphocytosis.

In *congenital syphilis* there is always anæmia. The hæmaglobin is reduced to the lowest figure. There is a great diminution in the reds, nucleated reds are seen in abundance, and the white cells, especially the lymphocytes, are increased. Loos reports a fatal case in which the leucocytes reached 58,000.[1] Cabot reports a case of syphilis with leucocytosis of 100,000, and Monti, Baginsky, and others have also reported excessive leucocytosis. If the skin lesions are severe there may be an excess of eosinophiles. The spleen is usually decidedly enlarged.

In *rickets* there is usually a simple chlorotic anæmia. The leucocytes may rise to 30,000. (Luzet, however, believes that all grades of leucocytosis, even to a true leukæmia, exist in rickets, and there is a decided belief among many observers that both syphilis and rickets not infrequently lead to leukæmia; a lymphocytosis is usually the result.) In complicated cases the blood picture becomes not unlike that of v. Jaksch's disease—that is, there are unusual numbers of nucleated reds with

[1] Ewing, p. 297.

many poikilocytes. In such cases Ewing states the hæmaglobin index is unusually *high*. The spleen in these cases is enlarged, and there are usually other evidences of rickets, such as the beading of the ribs and changes in the other bony structures.

In *v. Jaksch's disease*, which is supposed to be peculiar to infants and young children, the following changes in the blood are considered characteristic: There is a great and unusual diminution in the number of red corpuscles. There is always a leucocytosis, which, however, is not so great or progressive as in leukæmia. There is a remarkable variety of the forms and sizes of the leucocytes. The reds show great poikilocytosis; there are numerous nucleated reds to be seen. The clinical characteristics of the disease are the great enlargement of the spleen without marked enlargement of the liver, the edge of which is sharp, and without marked glandular enlargement. The prognosis is relatively good. Three cases which came to post-mortem presented no evidences of leukæmic infiltration. The disease has an anatomical basis, though Monti and Buggrum, in analyzing twenty cases, found evidences of rickets and syphilis in sixteen. It is also to be remembered that prior to 1892 (v. Jaksch's disease was described in 1889) there is no case on record, according to Fischl, where a differential count was made.

Splenic Anæmia. A disease not common in children, though recently numerous observers have reported cases in infants. In this condition the blood shows no pathognomonic changes. There is usually but little change in the form and size of the red corpuscles, while the hæmoglobin is much reduced—a chlorotic anæmia. There is usually but little or no increase in the leucocytes unless high temperature has been associated with the disease; indeed, in Osler's fifteen cases—all adults—a leukopænia was the rule.[1] Nucleated reds are rather scanty, and may not be found at all. The spleen is usually enlarged and tender; this enlargement precedes the anæmia,[2] the liver is often slightly enlarged, while there is sometimes slight jaundice present. The digestive symptoms are probably the most prominent. There is a tendency to hemorrhage either from the stomach or bowels, not infrequently from the nose. The duration of the disease is protracted, its cause is unknown, recovery has been seen.

Leukæmia. A disease which is rather uncommon in infancy, though the literature now contains quite numerous reports of cases occurring from one month up to extreme old age. For purposes of brevity we can assume an acute and chronic variety. The chronic variety, or myelogenous type, is the more frequent. The blood picture

[1] The American Journal of the Medical Sciences, January, 1900, p. 54.
[2] Osler. Ibid.

is typical. The leucocytes are increased to astounding proportions, sometimes one to two, four or six. A large proportion of these cells are myelocytes; mast cells are present in considerable quantities, as are also nucleated reds; the mast cells Ewing considered almost pathognomonic. Eosinophilic myelocytes are numerous. The disease is chronic in its duration, we do not know its cause, and cure is almost unknown.

Lowit has described a protozoon, and many others have found similar bodies in leukæmic blood, but no one has been able to verify or follow up his findings. The lymphatic variety is more rare, and the acute cases are usually of this type, the majority of these cases occurring before the age of twenty-five. Thus, Muir states that of the seventeen cases reported by Ebstein, but seven were over thirty, and Muir's four were all under twenty-six. The blood picture is here almost characteristic. The leucocytosis is usually about 200,000. A large proportion, 85 per cent. to 95 per cent. of them, are lymphocytes. Myelocytes are found but in very scanty numbers. Nucleated reds are also extremely scarce, but are sometimes found. Segmenting lymphocytes and the nuclei of large lymphocytes, without cytoplasm, are frequently seen. Vacuolated and degenerated or disintegrating leucocytes are frequent. There is no marked reduction of the red corpuscles, nor is there much change in size or shape. The hæmoglobin keeps pace with the reds. The clinical symptoms depend to some slight extent upon the variety of the disease. In the spleno-myelogenous form the onset is gradual; epistaxis is a more common symptom; diarrhœa is usual; there is increasing weakness, a peculiar grayish pallor, with decided splenic enlargement. The latter is often noticed by the patient himself. In the lymphatic variety there is usually enlargement of the entire superficial lymphatic apparatus. The glands are rather elastic, but become soft. At times the superficial glands are not affected, though the blood picture remains the same as before stated. Under such cases there is usually a leukæmic infiltration of either the glands along the intestinal canal or in the viscera. The spleen is not usually affected, though sometimes it is slightly enlarged. Thus Muir[1] cites a recent case of his own in which the liver, kidneys, and adrenals were infiltrated, while the glands were almost unaffected. Lymphatic leukæmia, therefore, in infants is not common. Morse[2] gathers twenty cases of leukæmia; but one of these, however, was accompanied with a differential count, and as the diagnosis in some of the cases was made by clinical signs, without study of the blood, many of these cases must be thrown out. Morse's case was of mixed variety. A. E. Taylor in his studies on leukæmia[3] reports six-

[1] Allbutt's System, vol. vi. p. 656.
[2] Boston Medical and Surgical Journal, August 9, 1894.
[3] Pepper. Clinical Laboratory, vol. ii. p. 148.

teen cases of leukæmia, four of which are lymphatic, but none in patients under eleven years of age. Morse, again in 1898,[1] quotes seven acute cases in children from the literature, and describes his own case, in which but one blood examination was made. McCrae[2] reports an acute case of leukæmia occurring in a boy of three years, and includes thirteen previous cases from literature. Ewing (page 201) states that Audeod has observed leukæmia in fifty-six children; eleven times in the first year, twelve times from the second to the fourth year, twelve times from the fifth to the ninth year, and twenty-one times from the tenth to the fifteenth year.[3]

The blood picture in my own case corresponds accurately to the description given of pseudoleukæmia anæmia infantum, or v. Jaksch's disease, and the clinical picture also coincides with its description. In none of the cases, however, of v. Jaksch's disease have I been able to find a careful report with differential counting of the blood, and many of the cases reported as such have been based upon the presence of a leucocytosis, sometimes quite small, with evidences of anæmia and decided enlargement of the spleen. The differential counting of the leucocytes, it seems, must be practised in these cases before an accurate diagnosis can be made. This fact leads me to this query: "Does our knowledge of the blood at the present day allow us to acknowledge that the disease described by v. Jaksch is an entity?" If it is not, in what group of diseases can it be placed?

In order to discuss this question, let us briefly consider the effect upon the blood: First, of simple splenic enlargement. Second, removal of the spleen: (*a*) for injury, (*b*) in splenic anæmia, (*c*) in myelogenous leukæmia. Simple splenomegaly in acute diseases, such as typhoid fever, malaria, etc., is accompanied by a secondary anæmia, which is probably induced by the disease. The leucocytes are usually diminished in number (leukopænia). In the more chronic enlargements, such as malaria or splenic anæmia, there is again a leukopænia, or a mild grade of leucocytosis, as in rickets and syphilis; thus the most marked effect upon the blood seems to be a relative reduction in leucocytes. But this is relative, for with the onset of complications a decided leucocytosis takes place, showing that the leucocytes are present in the body, though not in the peripheral blood. This leukopænia I have also seen in cirrhosis of the liver, accompanied with marked ascites and splenic enlargement, and also in another case of simple splenomegaly whose blood showed an intense chloro-anæmia. This latter case was classed as a splenic anæmia for want of better diagnosis, though he presented none of the ordinary symptoms of the disease.

[1] Archives of Pediatrics, 1898, vol. xv. p. 130.
[2] Johns Hopkins Bulletin, May, 1900, p. 102.
[3] Traite de Malade d'Enfance, t. ii., p. 112.

Bovaird has reported two cases of chronically enlarged spleen with the pathological diagnosis of endothelioma, in which the blood changes were but slight. In the first case, a child, aged three years, the erythrocytes numbered 4,400,000; the leucocytes 9000. In the second case, a child, aged thirteen years, the erythrocytes numbered 2,880,000, and the leucocytes 4000.

Sailer, in a recent paper, has reported six cases of enlargement of the liver and spleen—with enlargement of the lymph glands—in which the presence of tubercle bacilli was demonstrated; in these cases the blood showed no special changes, there was no leucocytosis, and the differential count was normal.

The total removal of the spleen has a decided effect upon the blood. In comparatively healthy subjects Ewing states (page 241) that the spleen may be removed without affecting the blood more than any other laparotomy. Such an operation is usually followed by a leucocytosis ranging from 15 to 50,000, which may persist from two to six weeks, or even longer. In traumatic cases the blood may resemble an acute leukæmia, although this does not always follow. The spleen has also been removed for diseases such as malaria (ague cake), splenic anæmia (Osler only so advises when hemorrhages are recurrent), and myelogenous leukæmia. In the latter the result usually is fatal, and no blood counts of value, subsequent to the operation, are to be found. Warren, however, has included in his article on "Surgery of the Spleen," a case of myelogenous leukæmia, operated upon by Richardson, with recovery.

In splenic anæmia the results of operation are better. Osler, Warren, and Malcolm L. Harris, with Max Herzog, and others have reported cases of removal of the spleen for this disease. In Warren's case the leucocytes after operation remained up for three months, the highest count being 24,000. Harris and Herzog's two cases showed a leucocytosis of 28,000 before operation, with no subsequent increase after the removal of the spleen. They noted, however, a great increase in the eosinophiles, after four months to 14.4 per cent. This change in the differential count is said not to occur until some months subsequent to the operation. They also noted a decided increase in the mononuclear cells.

For malaria the spleen has frequently been removed. Hogan quotes sixty-four cases since 1890, with a mortality of 23.4 per cent. No statements are to be found as to its effect upon the blood. From the above it can be seen, therefore, that no one effect upon the blood is produced by simple enlargement of the spleen, or by its removal in health or in disease. Observers differ as to whether splenic enlargement precedes, as in splenic anæmia, the blood condition, or whether the latter produces the splenomegaly. In splenic anæmia Dr. Osler considers the

enlargement antedates the anæmia, and is not its cause. Harris and Herzog consider the splenomegaly the cause of the anæmia, and call attention to the good results of operation to support their contention.

In the case above considered there was present a typical clinical picture of chronic splenomegaly, with a leucocytosis of a slowly increasing lymphatic type; the presence of nucleated reds of all sizes and ages, degenerative changes in the reds, the same changes in the leucocytes, the presence of a few myelocytes, with death from asthenia, and, alas, no autopsy. That these cases exist without leukæmic infiltration of tissues is true, for in two cases coming to autopsy, Ewing has failed to find any such pathological change. The duration, the course, the blood picture, and the termination of this case are in common with the leukæmias of adult life; at the present time one cannot predict how long the leukæmias will be permitted to remain in the class of primary or essential anæmias: they present many of the characteristics of true secondary anæmias, and not a few observers consider that in the secondary anæmias, splenic anæmia and pseudoleukæmia anæmia infantum have their proper position. I am inclined to support this view.

[NOTE.—Since writing this article there has been published in the *Boston Medical and Surgical Journal* a comprehensive and elaborate article on the "Association of Anæmia with Chronic Enlargement of the Spleen," by Arthur H. Wentworth (concluded in the number of October 31, 1901). The conclusions reached in the article are valuable, while the entire literature from 1879 to 1900 is appended. Stengel, as early as 1896, in his article in the *Twentieth Century Practice*, contended that pseudoleukæmia anæmia infantum was not an independent disease.]

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UNCINARIOSIS (ANKYLOSTOMIASIS). A FURTHER REPORT OF A CASE, WITH NOTES UPON THE AUTOPSY.[1]

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In view of the fact that a number of cases of uncinariosis have been discovered in the United States (Missouri, New York, Louisiana, District of Columbia, Texas, Virginia, Maryland—not all yet reported) in the last few years, the majority in the past few months, and since, to many of us, unless engaged in teaching, it is little more than a name, it seems advisable to bring this serious disease to the attention of the profession in some detail.

It is my belief, from the nature of the disease, as well as from recent developments, that it is far more common among native Americans than has been the general impression heretofore. Besides, there is the ever-increasing danger of its importation in emigrants from localities where the disease is prevalent, and in those returning from our newly acquired possessions.

Uncinariosis (ankylostomiasis) is a chronic disease due to infection by a small thread-worm known as the uncinaria duodenalis (ankylostoma duodenalis), which is characterized clinically by intense anæmia, dyspnœa, weakness, gastro-intestinal disturbances, and, in advanced cases, by œdema of the face and extremities and serous effusions into the cavities of the body.

History. The disease has been described under various names: tropical chlorosis, Egyptian chlorosis, geophagia, mal d'estomach, cachexie aqueuse, cachexia africana, tunnel disease, miner's disease, etc.

In the *Papyros Ebers* (German translation by Dr. H. Joachim), the oldest book upon the subject of medicine, written in the middle of the sixteenth century before Christ, is recorded a disease among the Egyptians known as the ā a ā, which so strongly resembles uncinariosis in its symptomatology that it seems at least possible that they are identical. The similarity is further strengthened by the mention of remedies given for worms in the intestines of those afflicted with this disorder.

From the fact that it is several times spoken of as the deadly ā a ā, it must have been very fatal in those ancient times.

According to Sandwith[2] the first mention of the disease in modern times was in Brazil in 1648, and in the West Indies and in

[1] Read before the Medical Society of the District of Columbia, November 6, 1901.
[2] Observations on Four Hundred Cases of Ankylostomiasis, Lancet, 1894, vol. i.

Guiana in the early part of the eighteenth century, while in Europe it was first noted among the miners of Auzin in 1802.

To Angelo Dubini, of Milan, is due the credit of discovering the nematode in man which is the cause of the disease. He, in 1838 (published 1843), found the parasite[1] in the small intestine of a woman dead of pneumonia; but the importance of the discovery was not realized until 1854, when W. Griesinger declared, upon the evidence of a single autopsy, performed in Cairo, April 17, 1852, that Egyptian chlorosis was caused by the ankylostoma duodenalis.[2] This fact was apparently lost sight of until, in 1866, it was confirmed by Wucherer.[3]

The disease came into prominence in Europe in 1879 or 1880, when a number of workmen in the St. Gothard tunnel developed a severe form of anæmia, which was found to be uncinariosis.

The geographical distribution is wide, the disease having been reported from most of the countries of Europe, in Africa (especially Egypt), Australia, South America, the Indies, the United States, and other localities. As would be expected, it is more common in the tropical and subtropical countries. Sandwith states that the parasite has been found between the parallels of 52° N. and 30° S., but that above 47° N. it can develop only in sheltered localities, such as mines, tunnels, etc.

As our possessions, excepting Alaska, lie entirely below the 50th parallel, there seems to be no reason why we should not meet with uncinariosis more frequently than we have heretofore.

I am told by Dr. Stiles that parasites of the same genus as that found in man cause the death of many dogs in this locality, and are very common in Alaska in the fur-bearing seal, and through the South in sheep, goats, and cattle.

There is thus far no proof, however, that these parasites are transmissible to man.

According to Manson, in Egypt the parasite is found in nearly every cadaver, and in India it was recovered from 75.58 per cent. of 1249 natives to whom thymol had been administered.

It must not be supposed, however, that all those who are infected show the symptoms of uncinariosis; far from it, many are strong and healthy. The weak and poorly nourished are those who are least able to withstand the loss of blood occasioned by the parasite.

PARASITE. The worm is nearly cylindrical in shape, from 0.4 to 1 mm. ($\frac{1}{60}$ to $\frac{1}{25}$ inch) in diameter, and from 6 to 18 mm. ($\frac{1}{4}$ to $\frac{3}{4}$ inch) in length. It is yellowish or grayish-white in color, with translucent edges. The males are more slender and more transparent than the

[1] Dubini gave the name of ankylostoma duodenalis to the worm, which, according to zoölogists, is wrong, since the genus is identical with that found in the wolf, in 1789, by Frölich, and called by him uncinaria.

[2] Archiv für Physiologische Heilkunde, 1853-1854.

[3] Gazeta Medica da Bahia, 1866, and Archiv f. klin. Med., 1872.

females by reason of the less extensively developed sexual organs. The head end is always bent backward. The tail, which can be distinguished by the swelling at the end (the bursa copulatrix) is much more curved than the head, sometimes appearing even to be rolled up. The length of the mature male is from 6 to 11.5 mm. The diameter is nearly the same for the posterior two-thirds (about 0.5 mm.), but the anterior third tapers down and ends in a truncated cone at the head. The female is usually larger (from 6 to 15 mm.), much thicker (about 1 mm.), and is generally found slightly curved dorsally. The number of the females is, as a rule, much larger than that of the males.[1] The armamentarium of the parasite consists in four claw-like hooks set in the margin of the mouth capsule, two on either side of the ventral line, and two conical teeth, one on each side of the dorsal line. By means of these the worm clings to the mucous membrane of the small intestine and sucks the blood from its host.

From the fact that the blood cells are found unchanged in the parasite, it is believed that only the serum is used for its nourishment; thus it becomes a most wasteful feeder.

The ova, which are a perfect oval in shape, measure 0.055 to 0.065 mm. by 0.032 to 0.043 mm., and are covered with a transparent shell through which may be seen the segmented yolk.

Ova of the uncinaria duodenalis (multiplied about 300 times).

Looss[2] has proved by experiment that the development of the egg depends upon the surrounding temperature, its accessibility to the air, the amount of moisture, and upon the character of the culture media. Thus he found that in Alexandria, Egypt, where the temperature in summer varies around 80° F., the eggs on the surface of the media develop and the embryos escape in twenty-four hours. If a cool night intervenes, with a temperature of from 70° F. to 72° F., these changes do not occur for thirty-six to forty hours, and that a temperature of 33.8° F. kills the eggs in twenty-four to forty-eight hours. He also found that too much moisture not only delayed the development of the eggs, but caused the death of a large number of them.

About the second or third day the rhabditiform embryo, when under favorable circumstances, undergoes the first ecdysis—*i. e.*, casts its cuticle, which has by this time become loosened. From the fifth day

[1] J. Ch. Huber. Twentieth Century Practice of Medicine.
[2] Centralbl. f. Bakter. u. Parasitenkunde, 1896.

on the second ecdysis takes place. The cuticle is seen to divide into two layers, which separate more and more until the outer cuticle is separated from the body, but remaining around it, thus giving rise to the theory of encystment.

The young worm has now reached the infection stage and is ready to enter its host and begin its parasitic life. Looss has succeeded in keeping worms alive in this condition for three months. The ripe embryo prefers water as its medium, and may thus easily be taken into the system. He has shown that both eggs and embryos are killed by being dried out, which tends to disprove the theory of infection through dust which may be blown about.

Looss[1] estimates that about four or five weeks are necessary after infection for the worm to become fully mature, and that in Egypt five or six weeks is the probable period necessary for the freshly laid eggs to develop into individuals containing eggs.

As there is no development of the eggs in the host it follows that any new infection must come from outside. This seems a very fortunate circumstance, as unless the primary infection is a large one, or the exposure is repeated, there will be no ill effects in the majority of instances.

The parasites, which sometimes number thousands, are said to be found in greatest abundance in the jejunum, secondly in the duodenum, and to a lesser extent in the ileum. This, however, was not my experience (see autopsy). The young worms change their positions frequently, thus allowing a large amount of blood to escape from the wounds deserted by them for new fields.

Mode of Infection. The embryo finds its most favorable habitat in damp soil, hence the vast majority of those infected are either earth eaters or are engaged in work which brings their hands in contact with the earth. Thus miners, gardeners, workers in brickyards, etc., are especially subject to the disease. Sandwith found in 200 cases carefully studied that 190 worked in more or less damp soil with their hands. These people probably convey the infection to their mouths by eating without having previously cleansed their hands. Some, no doubt, are infected through drinking-water, but Giles' experience does not bear out this theory. He examined fifty-six water-supplies of infected villages, and found one doubtful embryo.

F. M. Sandwith[2] rather supports the idea suggested by Looss, that infection may take place by the penetration of the embryo through the skin, and quotes an experience of that author as follows: Looss accidentally let a drop of water which contained numerous embryos fall upon his hand. Noticing that the spot became red and burned, he let

[1] Centralbl. f. Bakter. u. Parasitenkunde, 1897, p. 913.
[2] British Medical Journal, September 14, 1901.

fall another drop, which produced the same result. On careful examination of the area he found countless sheaths which had been discarded by the embryos, indicating that they had thrown off their outer covering and entered the skin. Shortly thereafter he developed symptoms of uncinariosis and found ova in his evacuations. This, however, was not positive proof that he was infected at this time, as he had previously suffered from the disease. Looss then tried several experiments to prove that the embryos could penetrate the skin, one of which was successful. He allowed a drop of infected water to fall on the leg of a boy who was to have the limb immediately amputated. That portion of skin was then hardened, and sections made which showed that the embryos had entered the skin by way of the hair follicles.

Whether the embryos are able, after penetrating the skin, to reach the intestinal tract remains to be proved.

Morbid Anatomy. "The pathological anatomy is that of extreme anæmia plus the local lesions in the small intestine due to the presence of the uncinaria." There is general bloodlessness of all the tissues; œdema and effusions into the cavities; the fat is usually fairly well preserved; the heart is enlarged, dilated, and may have undergone fatty change; the spleen and kidneys are very often lardaceous and waxy, or the spleen, and still more frequently the liver, presents a certain degree of atrophy. If the autopsy is performed a short time after death the parasites may be seen clinging by their mouths to the mucous membrane of the small intestines. If the body has grown cold they are found in the mucus.

The intestinal wall may be thickened from a catarrhal process. Scattered about the intestine in and under the mucous layer, and visible through the serous and muscular coats, may be seen small ecchymotic spots which, on close inspection, show minute punctures indicating the points where the parasites are attached.

In a few instances parasites have been found in small cavities filled with blood under the mucous membrane. The cavities communicate with the interior of the intestine by small punctures through which the worms have gained an entrance. The intestinal contents may be streaked with blood, and very occasionally there may be extensive hemorrhage.

The intense anæmia is probably due, first, to the actual abstraction and leakage of blood from the mucous membrane of the intestine; secondly, to interference with the proper digestion and absorption of food because of a catarrhal process set up in the intestine by the bites of the parasite; and, lastly, it has been suggested that there is an absorption of poisons produced by the parasite itself or from the intestinal contents through the wounds made in the mucous membrane which aids in causing the anæmia.

There is apparently no actual hæmolysis, as the amount of iron in the liver is not increased, as is the case in pernicious anæmia.

SYMPTOMS. According to Manson[1] "the essential symptoms are those of a progressive anæmia, an anæmia which is usually associated with dyspeptic trouble, but which, in uncomplicated cases, is not associated with wasting. If the progress of the case is unchecked serous effusions in different organs and fatty degeneration of the heart ensue, and death may occur from syncope or intercurrent complications." There may be either diarrhœa or constipation, or the bowels may be regular. Occasionally the evacuations have a reddish-brown shade, due to the admixture of blood, and very rarely there may be distinct hemorrhage from the intestine. The appetite is variable; it may be excessive, capricious, or lost entirely. In advanced cases there is dyspnœa on slight exertion, with palpitation, œdematous swelling of the face, feet, and ankles, and extreme weakness. The urine is usually alkaline or neutral, with a specific gravity of 1010 to 1015; a trace of albumin may be noted in advanced cases. There is often stupidity so dense as to amount, in some cases, almost to weak-mindedness. If a severe infection occurs before puberty that period is apt to be delayed. The temperature is said, as a rule, to be subnormal, though there may be transient flashes of fever. The heart is usually enlarged, and hæmic murmurs are to be heard at the base and over the vessels of the neck. As a rule, there is no enlargement of either liver or spleen. The blood shows the following changes: A marked reduction in the red cells, sometimes below 1,000,000; a proportionate reduction in the hæmoglobin, poikilocytes, and nucleated red cells. Some observers have noted megaloblasts as well as normoblasts.

Of nineteen cases studied in Porto Rico by B. K. Ashford,[2] fourteen showed normoblasts and seven megaloblasts. The megaloblasts were, however, never in excess of the normoblasts (an excess of megaloblasts is considered quite characteristic of pernicious anæmia).

There is no leucocytosis in uncomplicated cases. Ashford noted an excess of eosinophiles in several cases.

DIAGNOSIS. It has very aptly been said that the way to make a diagnosis of uncinariosis is "first to suspect its presence." Once having had the suspicion aroused by the peculiar anxious expression of the face and the intensely anæmic appearance of the patient, it is a very simple matter to examine the stools for the ova, which are present usually in large numbers. The parasites themselves are almost never seen until after the administration of an anthelmintic.

Both ova and parasites are readily recognized from the cuts in works on clinical diagnosis.

[1] Tropical Diseases.

[2] New York Medical Journal, 1900.

If a microscopist is not at hand, the administration of thymol to suspects would cause the parasites to appear in the stools, where they may be seen with the naked eye.

The disease bearing the closest resemblance to uncinariosis is pernicious anæmia. The similarity is most striking in the insidious onset, pallor, gastro-intestinal disturbances, circulatory symptoms, weakness, dyspnœa, œdema, tendency to periods of improvement followed by relapses, marked reduction in the number of erythrocytes, presence of poikilocytosis and nucleated red cells, and absence of leucocytosis.

It is said that in uncinariosis the color index is usually low, while in pernicious anæmia it is high—*i. e.*, there is a greater proportional reduction in hæmoglobin in uncinariosis; but this is not always the case. In fact, it is many times almost impossible to differentiate between the two diseases without an examination of the evacuations to determine the presence or absence of the ova.

In leukæmia the blood is very different, as a rule, though similarity has been reported.

True chlorosis is readily differentiated by examinations of the blood and feces; malarial cachexia by the enlarged spleen, presence of plasmodia and absence of the ova of the uncinaria.

In every case of anæmia not attributable to some definite cause the evacuations should be examined microscopically for the ova of the uncinaria.

Prognosis. If the diagnosis is made and proper treatment instituted before the disease has existed long enough to produce serious changes in the tissues and organs of the victim, recovery is prompt; but if the condition goes unrecognized for months or years the result is not infrequently fatal. A small number of cases are very acute and severe, death resulting in a few weeks after the first appearance of symptoms. As a rule, however, the course extends over some years.

Of Sandwith's 400 cases, 89.5 per cent. were cured, or greatly improved; 2.5 per cent. were unimproved, and 8 per cent. died

In prolonged cases there are usually periods of temporary improvement.

Even if death does not result directly from uncinariosis, those afflicted are especially prone to succumb to some intercurrent disease.

Treatment. The recognized treatment is thymol administered as follows: 15 to 30 grains repeated four times at intervals of one and one-half to two hours. If the bowels do not act spontaneously in twelve hours after the last dose, give a purgative, which is usually not necessary. It is well to clear the bowels the day before with a purgative, and place the patient on liquid diet. The fact that thymol is poisonous must not be lost sight of, and the patient should be carefully watched. Since the drug is freely soluble in alcohol, ether, turpentine, chloroform,

oil, glycerin, and certain alkaline solutions, these should be withheld during its administration.

In seven or eight days the stools should again be examined, and if the ova are still present the same programme should be carried out and repeated until all the parasites have been destroyed.

The treatment of the anæmia from uncinariosis is the same as in that secondary to any other cause.

History in the United States. It seems very probable that the form of anæmia spoken of by Jean Louis Chabert (1821), James B. Duncan (1850), and others, as existing to a considerable extent in our Southern States was uncinariosis.

The number of authentic cases, however (in which the diagnosis was established by the finding of the ova), reported by observers in the United States is as yet small, and, as far as I have been able to learn, appeared in the following order:

The first by W. L. Blickhahn,[1] of St. Louis (1893); five cases by F. G. Möhlan[2] (1896); one case by C. H. Tebault, Jr.[3] (1899); one by J. H. Dyer[4] (March, 1901); my own case[5] (June, 1901); Allyn's case[6] (July, 1901); one case by Wm. B. Gray[7] (September, 1901); and one by M. Charlotte Schaefer,[8] from Texas (October 26, 1901), making twelve in all. Of these twelve cases, five undoubtedly had their origin in the United States; three probably brought the infection from abroad, one from Mexico having been in the United States only a short time when symptoms appeared; in three the data upon which to base an opinion upon this point was not given.

Dr. Gray also reports a second case, a sister of the one noted above, but as neither ova nor parasites were found it does not seem justifiable to include it among the authentic cases.

My friend Dr. Allen J. Smith, of the University of Texas, writes me that he is inclined to believe that the disease is quite common in Texas, since he found the ova in the evacuations of eight out of eighty-eight students. The majority of these cases, Dr. Smith says, have never been outside of the State. His forthcoming paper upon his observations will be very interesting.

G. R., American, aged nineteen years, of Westmoreland County, Va., occupation, light farm work, was admitted to the Garfield Hospital, of Washington, D. C., May 31, 1901. The patient was a small baby at birth, but was said to have developed normally until about three years previous to his admission. This, however, was prob-

[1] Medical News, December 9, 1893.
[2] Buffalo Medical Journal, 1896-1897.
[3] New Orleans Medical and Surgical Journal, 1899-1900.
[4] Interstate Medical Journal, St. Louis, March, 1901.
[5] Philadelphia Medical Journal, June 29, 1901.
[6] American Medicine, July 13, 1901.
[7] Virginia Medical Semi-Monthly, September 27, 1901.
[8] Medical News, October 26, 1901.

ably a mistake on the part of his friends, as the boy's development was that of a lad under fourteen. He was small, childish in voice and manner, and the genital organs were infantile. His mentality was much below the average; in fact, he seemed very stupid and dull. He had never had any of the diseases of childhood, nor had he had malaria. About three years prior to his admission it was noticed that the boy was pale; one year later the anæmia became very marked, and there was swelling of the face, abdomen, and feet. There was, however, an improvement until about three months before he came to the hospital.

The chief symptoms were great weakness, dyspnœa on the slightest exertion, and pallor. Contrary to the rule in these cases, there was no complaint of any gastro-intestinal disturbance.

The expression was anxious and dejected. The skin was of a faint yellow tint; the face was bloated, especially about the eyes.

The mucous membranes were extremely pale, the body well nourished, but the muscles flabby. The tongue was clean. The heart's impulse was widely diffused, the apex-beat was in the fifth interspace and almost as far to the left as the mid-axillary line. Over the base there was a loud systolic murmur, the point of maximum intensity being in the pulmonary area. This murmur was also heard in the vessels of the neck. The second pulmonary sound was accentuated. At the apex was heard a systolic murmur which was transmitted to the axilla and to the angle of the left scapula. A faint diastolic murmur was also present over a small area near the apex. The feet and ankles were œdematous. The urine was clear, pale, acid, and showed a specific gravity of 1010. The lowest blood count, June 4th, showed the red cells to be 1,450,000 per cubic millimetre (29 per cent. of normal), the whites 4400 (slightly below the average), hæmoglobin 30 per cent. Dr. J. B. Nichols' report upon the stained cover-slips, June 9th, is as follows: Macrocytes and microcytes a few; polychromatophilia, not observed; poikilocytes, a few; microblasts, normoblasts, and megaloblasts, none.

Leucocytes: Small mononuclear, 15 per cent.; large transitional, 9 per cent.; polynuclear, 71 per cent.; eosinophiles, 5 per cent.; microscopical examination of the evacuations showed innumerable ova of the uncinaria, upon which the diagnosis of uncinariosis was made, and later confirmed by Dr. C. W. Stiles, of the Department of Agriculture, who very kindly saw the case with me and gave valuable advice as to the recovery of the parasites, etc. No Charcot-Leyden crystals were observed in the evacuations, which is of interest in connection with the absence of eosinophilia.

During the early treatment of the case an attempt was made to count all the worms passed; this, however, was abandoned after two weeks, as the number had then run up to 407, and the supply seemed inexhaustible in spite of weekly administrations of thymol in two doses of 30 grains, separated by an interval of two hours and followed by a purgative.

The ova were looked for each week, and gradually decreased, but up to the time of the patient's death, from cerebral hemorrhage, two months after admission, never wholly disappeared.

The general condition improved, but there was always fever, the temperature ranging between 99° and 100°. This continued fever is contrary to the rule.

The last blood count was on August 3, the day before his death, and showed a marked improvement. Reds, 3,033,000 (60 per cent. of normal); whites, 3200; hæmoglobin, 52 per cent.

The appetite was fairly good, the weight had increased, and there seemed to be every reason to hope for a favorable outcome, when, upon August 4, the patient fell unconscious with the symptoms of cerebral hemorrhage, which shortly proved fatal. A similar fate to that of one of Sandwith's cases.

One point which seems worthy of note is the remarkable resistance to treatment shown by the worms. In spite of weekly administrations of thymol in large doses for a period of two months, there were still a few parasites left.

The autopsy was performed by Dr. Nichols.

Necropsy. Body fat and well nourished, but pale. Genitals infantile. The small intestine was of large calibre and had thick walls, especially the duodenum and jejunum. There were no perceptible induration, cicatricial markings, or contractions of the intestinal walls. The valvulæ conniventes were very prominent, and along their bases were rows of very minute points of congestion or hemorrhage; aside from these doubtful points, no distinct hemorrhagic areas, obvious macroscopically, were present. The mucous coat of the bowel was lined with viscid chyme or mucus in which were embedded some uncinaria which were in relatively small numbers. Twenty-nine of the worms were found distributed indifferently along the entire small intestine. A few others may have escaped notice. One of the worms was found with its head attached to the mucosa of the ileum.

The descending colon was studded with small round gray points, apparently enlarged solitary follicles. The mesenteric glands were enlarged. The liver was much enlarged, weighing 2390 grammes; firm, pale in places. The pancreas was firm and hard. The spleen much enlarged, 17x11x5 cm. in size, weighing 410 grammes; congested, fairly firm, and mottled.

The kidney of horseshoe variety, the two organs being united at the lower end; much enlarged, the whole weighing 455 grammes. There was one ureter on each side, both passing down in front of the conjoined kidney. The bladder was distended with urine. The pericardial sac contained a noticeable amount of serum. The heart was much enlarged, weighing 467 grammes. The wall of the left ventricle thickened, the cavity nearly empty, the right containing soft, post-mortem clot. Valves normal. Foramen ovale closed. The left lung much contracted and retracted back into the thoracic cavity, with extensive pleural adhesions easily broken up; weight 273 grammes. Right lung normal, weight 273 grammes.

The brain showed a general subdural hemorrhage over the left cerebrum and a subdural hemorrhage on the right side over the upper portion of the fissure of Rolando. On the right side there was softening of the cerebral substance about the upper end of the fissure of Rolando, giving rise to hemorrhage, the extravasation of blood extending outward under the dura. In the left cerebrum there was similar softening in the occipital lobes with hemorrhage extravasating under the dura. The ventricles contained no blood.

Cultures from liver and spleen were negative.

The only points wherein the pathological findings differed macroscopi-

cally from those usually observed in these cases were in the enlargement of the liver and spleen and in the scarcity of ecchymotic spots in the wall of the small intestine. The latter is readily accounted for by the fact that the parasites had nearly all been destroyed before the patient's death.

The microscopical observations, also made by Dr. J. B. Nichols, were as follows:

Specimens removed from various points in duodenum, jejunum, and ileum showed no material alterations. The subepithelial connective tissue of the muscosa was thickened, studded with lymphoid cells. The glandular epithelium was shrunken and apparently degenerated, but this was probably a post-mortem change. The epithelium cells of the glands of Brunner appeared enlarged and pale.

The colon showed enlargement of the solitary follicles with erosion of the overlying mucosa.

The mesenteric glands presented a normal appearance, the enlargement noted macroscopically being due to general hypertrophy of the gland structures. The spleen presented a normal appearance, without congestion or extravasation of blood; its enlargement consisting in a general hypertrophy. The heart-muscle was normal. The kidney showed congestion, bloodvessels and capillaries being filled with blood-cells. The parenchyma was practically normal, no fatty or amyloid change being observed. *The section from the liver was lost.* The only abnormalities noted were the tendencies to hyperplasia of the lymphatic structures.

LAPAROTOMY FOR PERFORATION IN TYPHOID FEVER.

REPORT OF SIX CASES.

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(From the Surgical Clinic of Dr. Dudley P. Allen, Lakeside Hospital.)

The report is based upon the six cases of laparotomy for intestinal perforation during the course of typhoid fever, occurring in Lakeside Hospital since its opening in January, 1898. These were performed in 1899 and 1900 during the writer's service as resident surgeon, and, with the exception of the last case, came under his immediate observation and care. The inclusion of this last case in the series is perhaps of doubtful propriety, since the patient entered the hospital as a case of general peritonitis and was treated as such, the case containing very little of interest with reference to the surgical treatment of typhoid perforation. For the purpose of completeness, however, it has been added.

It is due to the courtesy of the visiting staffs of Lakeside Hospital that the present report has been made possible, and to these gentlemen the writer wishes to acknowledge his obligation.

CASE I.—Male, aged forty-seven years. Moderately severe case. At entrance, leucocytes 5700 per cubic millimetre. Widal reaction negative, diazo positive. Bowels rather loose, no hemorrhage. Symptoms of perforation on the twenty-first day of the disease; abdominal pain, sensitiveness, rigidity, and moderate distention. Rise in pulse and temperature; slight vomiting; leucocytes 4460 per cubic millimetre. Condition unfavorable.

Operation. Five hours after appearance of symptoms. Chloroform. Time, thirty minutes. Right lateral incision. Dark-brown abdominal fluid. Perforation of ileum 8 centimetres from cæcum; closed with continuous Lembert catgut sutures. No threatening perforation found. Irrigation with sterile water; intestines not removed. Gauze drainage. Cultures from abdominal fluid, bacillus coli communis. Death seventy-five hours after operation, apparently from exhaustion.

Autopsy, restricted. Local peritonitis about lesion and gauze packing, catgut sutures absorbed, and perforation patent. Four other neighboring perforations opening into the same area of local peritonitis. Confluent ulcers of cæcum, not perforating. No general peritonitis. Cultures from local peritonitis, bacillus coli communis; from general peritoneal cavity, negative.

C. K., medical No. 769; surgical, No. 909. Service of Dr. H. H. Powell. Male, aged forty-seven years, white, German, married, carpenter. Admitted August 5, 1899; discharged August 15th. Dead.

Family History. Negative.

Previous History. Had always been well, strong, and active. Twelve years ago he had malarial fever; six years ago he had what was taken to be typhoid fever, being ill for nine weeks; no other illnesses recalled; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, loss of appetite, diarrhœa, and fever. Headache was the first symptom, beginning two weeks previous to entrance, and persisting. There was also anorexia and diarrhœa, the latter being quite troublesome. There was some abdominal pain in the region of the umbilicus.

Physical Examination. Patient well-nourished and developed; mind clear; face flushed; tongue coated. Pulse 80, good quality. Temperature 101.8.° Lungs negative. Heart not enlarged; soft systolic murmur at apex. Spleen enlarged on percussion, not palpable. Abdomen soft, slightly sensitive about umbilicus; numerous rose-spots. Leucocytes 5700 per cubic millimetre. Urine, no albumin; faint diazo reaction. Widal reaction negative.

During the next six days the symptoms were those of a moderately severe case of typhoid, the temperature running 101° to 103°, during which time he had six tub baths, four of which were given in the last two days. The bowels remained rather loose. The urine continued to show a diazo reaction, and there was a slight trace of albumin. No subsequent Widal tests were made.

On August 12th, the twenty-first day of the disease, the patient began to have abdominal pain, coming on rather gradually and being first noticed at 6.30 A.M. At first it was confined to the lower half of the abdomen, gradually spreading upward and becoming general. Pain was also noticed at end of penis. He vomited a slight amount of curdled milk at 8 A.M. The temperature at 6 A.M. was 101°, and at 9 A.M. had risen to 102.2°, and the pulse had risen from 72 to 104 during

the same period. The leucocytes at 9 A.M. were 4460 per cubic millimetre, and this unfortunately was the only count made. At 9.30 A.M. the patient was seen with Dr. W. H. Nevison. The abdomen was moderately distended and tympanitic. There was marked sensitiveness over the entire lower half of the abdomen, and the abdominal wall was generally quite rigid. Respirations 28, largely costal. Pulse was 124, dicrotic, readily compressible. The lungs were clear, the heart as previously described. The general condition of the patient was poor, although not altogether unfavorable for operation.

Operation, August 12th, 10.30 A.M., by Dr. W. H. Nevison, five hours after first appearance of symptoms. Anæsthetic, chloroform, preceded by morph. sulph., gr. one-quarter, hypodermically. Time, thirty minutes.

Light anæsthesia. Incision eight centimetres long in right linea semilunaris opposite anterior superior spine. Considerable dark-brown fluid escaped, fecal odor, containing small particles of extraneous matter thought to be feces. Cæcum readily found and ileum carefully inspected for about one hundred and twenty centimetres. A perforation was found about eight centimetres from the cæcum. It was round, about three millimetres in diameter, on the free side of the bowel, and from it fecal matter was escaping. The area of induration surrounding this opening was about two centimetres in diameter. No other perforations were found in the explored ileum or cæcum, nor were any perceptibly thinned areas appreciable, although careful search was made. The perforation was turned in without excision by a double row of continuous Lembert catgut sutures. The ileum was dark and congested, the cæcum somewhat so. Other coils of intestine seen were slightly congested. Appendix normal. Parietal peritoneum seemed but slightly congested. Presence of enlarged glands not observed. No fibrinous flakes seen. There were no adhesions. Abdominal cavity thoroughly irrigated with a large quantity of sterile water, special attention being paid to the pelvis and the lumbar fossæ. The intestines were not handled, but the irrigation was thoroughly accomplished by means of a hard-rubber flushing tube. Site of suture left near the surface, and to it iodoform gauze drainage was placed, brought out through the lower end of the wound. Above this the wound was closed with interrupted silkworm-gut sutures through all layers. During the operation the patient received strych. sulph., gr. one-fifteenth, hypodermically, and an infusion of seven hundred cubic centimetres of normal salt solution. The radial pulse was very rapid and weak, imperceptible at times. The operation was associated with considerable shock, the patient being in a poor condition on leaving the table.

The subsequent treatment of the case was by strychnine, nutrient stimulating enemata, liquids without milk by mouth, subcutaneous infusions of salt solution, sponge baths, and oxygen. Patient took nourishment well during entire time. He was slightly delirious and semi-conscious at times, but for the most part his mind was clear and active. There was slight abdominal distention, but practically no pain except on the day following the operation, when it was quite marked in the lower half of the abdomen for a short time. The discharge from the abdomen was very profuse during the last twenty-four hours, and was dark colored, which, together with the pain mentioned above, suggested strongly a second perforation in the locality of the gauze drain-

age. The temperature remained about 102.5° to 104° rectal, and the pulse 120 to 130. On August 15th, three days after the operation, the patient began to show evident signs of dissolution, the pulse-rate increased steadily, and the quality became progressively worse. There was some general abdominal distention, most marked in the epigastrium. There was an accumulation of coarse, moist râles throughout the chest, and the respirations were labored. Oxygen was given during the last seven hours. He was unconscious for six hours before death. He died August 15th, at 3.05 P.M., seventy-five hours after operation.

The bacteriological examination of the abdominal fluid at the time of the operation showed on cover-slip preparations a medium-sized bacillus and a small coccus, singly and in pairs. Plate cultures showed a pure growth of bacillus coli communis. No growth of the coccus mentioned could be found.

Autopsy. This unfortunately was restricted. About the gauze packing and the site of the lesion in the ileum there was a localized peritonitis, as was to be expected. Opening into this localized area was the original ulcer. The catgut suture had been almost entirely absorbed. There were four small neighboring perforations of the ileum, which also opened into this same localized area. There were several confluent ulcers of the cæcum which had not perforated. There was no general peritonitis. Cultures taken from the area of local peritonitis showed bacillus coli communis. Cultures and cover-slip preparations from general peritoneal cavity were negative.

CASE II.—Male, aged seventeen years; severe case. At entrance, leucocytes 5980 per cubic millimetre; Widal reaction negative; diazo reaction negative. Marked constipation; slight distention. Hemorrhage on seventeenth and eighteenth days of disease, preceded by considerable abdominal pain. Symptoms of perforation on eighteenth day of disease; abdominal pain, sensitiveness, rigidity, moderate distention, rise in pulse, temperature unaffected, slight vomiting. Condition very unfavorable.

Operation. Eight hours after appearance of symptoms. Chloroform. Time, twenty-five minutes. Right lateral incision. Dark-brown abdominal fluid with curds. Perforation of ileum twenty-five centimetres from cæcum; one threatening perforation close by; both secured with continuous Lembert silk sutures. Irrigation with salt solution and sterile water; intestines not removed. Gauze drainage. Cultures from abdominal fluid, bacillus coli communis. Death sixteen hours after operation; general peritonitis.

Autopsy, restricted. Intestinal suture intact. No other perforations found. General peritonitis. Cultures from general peritoneal cavity, bacillus coli communis.

J. B., medical, No. 779; surgical, No. 925. Service of Dr. J. H. Lowman. Male, aged seventeen years, white, American, single, student. Admitted August 13, 1899; discharged August 22d. Dead.

Family History. One brother died of typhoid fever; one sister ill with typhoid fever at present time.

Previous History. Children's diseases. Always strong and active; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, constipation, and general malaise. Headache was the first symptom, and was first noted ten days previously. Bowels constipated, no move-

ment for forty-eight hours before entrance. No hemorrhages. Chilly sensation during last two nights; no distinct rigor. Epistaxis on day of entrance.

Physical Examination. Patient well nourished and developed; athletic. Face flushed; tongue slightly coated, trembles when protruded. Pulse 116, low tension, dicrotic. Temperature 102.8°. Lungs negative. Heart slightly enlarged to the left, loud systolic murmur at apex. Spleen slightly enlarged on percussion, not palpable. Abdomen rather tense, not tympanitic, no especial sensitiveness, no rose-spots recorded. Leucocytes 5980 per cubic millimetre. Urine, no albumin, no diazo reaction. Widal reaction negative.

During the next four days the symptoms were those of a severe typhoid. The nervous symptoms were marked. There was a little abdominal pain on two occasions following tub baths. Baths were given every three hours with but few intermissions. Reactions following baths were not very good. The pulse ranged from 90 to 120, and was of rather low tension. On one day patient complained of considerable pain on inside of right leg, extending down to the knee.

On August 18th, at 8 P.M., following tub bath, there was sudden sharp pain in lower half of abdomen, which continued for some little time, but with rather less severity. The temperature was unaffected, but the pulse-rate was increased somewhat. Sponge baths were substituted. No blood examination was made. During the next day there was slight abdominal pain with an occasional increase in severity. The abdomen was somewhat distended, but this passed off after several free bowel movements. Following this the pain practically disappeared, and the patient was comfortable. There was no vomiting. Pain was present along inner side of right thigh. Leucocytes were 8000 per cubic millimetre in the morning, and 10,000 per cubic millimetre in the afternoon. The next day, August 20th, the abdominal pain had practically disappeared, but there was slight sensitiveness above the pubes and in the left inguinal region. Tub baths were resumed, and were borne rather better than previously. A small amount of old clotted blood was passed in one of the stools.

August 21st, the eighteenth day of the disease, the patient had been rather more comfortable than usual. At 2 A.M., while in tub bath, there was sudden severe pain in the lower abdomen. The abdomen was tense, not distended, and quite generally sensitive. The temperature was unaffected. The pulse rose from 80 to 110, and was not well sustained. At 2.45 A.M. the patient passed about two drachms of blood in a bowel movement. The patient vomited curdled milk at 3 A.M. Abdominal pain continued; sensitiveness was quite marked, more so in the right lower quadrant. Sponge baths were substituted. No blood examination was recorded. At 9 A.M. the patient was seen with Dr. W. H. Nevison. His general appearance was that of a very sick and much reduced man. The finger nails and lips were slightly cyanotic. Pulse was 120, regular, small volume, low tension. Respirations were 24, thoracic. The abdomen showed moderate general distention and tympanites. There was general sensitiveness, slightly more marked in the lower half, and marked muscular resistance. The condition of the patient seemed quite unfavorable for operation.

Operation, August 21st, 9.45 A.M., by Dr. W. H. Nevison, eight hours after perforation was thought to have occurred. Anæsthetic,

chloroform, preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty-five minutes.

Light anæsthesia. Incision in right linea semilunaris nine centimetres long, at level of anterior superior spine. A moderate amount of dark fluid containing particles of curdled milk escaped. Cæcum readily found, and ileum searched for about one metre from cæcum. About twenty-five centimetres from cæcum a perforation was found on the free margin of the gut, three millimetres in diameter; base generally thickened. About six centimetres from this thickened area a very thin centre was made out. Nothing more approaching perforation was found. Perforation was turned in without excision by two rows of running fine silk Lembert sutures for a distance of about two and five-tenths centimetres. The thin ulcer was also turned in with a single running silk suture. About the perforation was a little gray fibrinous deposit. The ileum had lost its gloss in places, but in general looked well. No fibrinous exudate outside the region of perforation. The cæcum and appendix normal. Remaining coils of intestines and parietal peritoneum appeared to be in good condition. No adhesions were found. Abdomen flushed with hot salt solution and sterile water in large quantities, special attention being paid to the dependent portions. This was done by means of a hard-rubber flushing tube. The intestines were not removed from the abdominal cavity. The site of suture left near wound, and to it was placed iodoform gauze drainage, brought out through lower end of wound, above which the incision was closed with interrupted silkworm-gut sutures through all layers. During operation patient's pulse became very feeble, respiration shallow, color poor. He was given strych. sulph., gr. one-fifteenth, hypodermically, divided, and a saline infusion of eight hundred cubic centimetres, under which the condition improved somewhat.

The subsequent treatment was by strychnine, nutrient stimulating enemata, subcutaneous infusions, and sponge baths. The reaction to the sponging was very marked, and the baths were given with considerable care. The patient's condition improved slightly for a few hours following the operation, after which time he failed steadily, and died August 22d, at 2 A.M., sixteen hours after the operation.

Bacteriological examination of the free abdominal fluid at operation showed a pure growth of bacillus coli communis on plate cultures. No cover-slip preparations were made.

Autopsy. This was very restricted. The intestinal suture was intact. No further perforation was found. There was a general peritonitis with thin turbid fluid. Cultures from the general peritoneal cavity showed bacillus coli communis; no cover-slip preparations were made.

CASE III.—Male, aged thirty-one years; mild case with a moderately severe relapse. At entrance leucocytes, 5700 per cubic millimetre; Widal reaction positive; diazo reaction negative. Bowels rather constipated; no hemorrhages. Symptoms of perforation on forty-third day of disease, twelfth day of relapse; severe abdominal pain, general sensitiveness, moderate distention, marked muscular rigidity on right side; rise in pulse, temperature unaffected; slight vomiting; leucocytes, 31,600 per cubic millimetre. Condition of shock, but fairly favorable for operation.

Operation. One hour after symptoms. Cocaine solution, 1 per cent. Time, twenty-five minutes. Right lateral incision. Little brownish

abdominal fluid. Perforation of ileum forty-five centimetres from cæcum, closed with double continuous Lembert silk suture; no threatening perforations found. Irrigation with sterile water, intestines not removed. Gauze and glass-tube drainage. Cultures from abdominal fluid showed staphylococcus pyogenes aureus. Death fifty-eight hours after operation; typhoidal toxæmia.

Autopsy, restricted. Sutures intact; no other perforation; no general peritonitis; no peritoneal adhesions except around drainage. Cultures from general peritoneal cavity, negative.

T. D., medical No. 992; surgical No. 1304½. Service of Dr. E. F. Cushing. Male, aged thirty-one years, white, Welsh, married, coachman. Admitted January 25, 1900; discharged February 28th. Dead.

Family History. Negative.

Previous History. Always strong and active, no definite diseases recalled; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, weakness, and lack of energy. Symptoms began ten days before entrance, with malaise. Following this there was severe headache, anorexia, slight cough, and marked weakness. He also had several attacks of abdominal pain described as "colic." The bowels were loose, but there had been no hemorrhages. Patient had vomited several times. There was no epistaxis.

Physical Examination. Well nourished and developed. Mind clear; tongue coated. Pulse 100, full, not dicrotic. Temperature 102°. Lungs and heart negative. Spleen enlarged on percussion, not palpable. Abdomen slightly distended and slightly sensitive on pressure; covered with scattering rose-spots. Leucocytes 5700 per cubic millimetre; hæmoglobin, 90 per cent. Urine, no albumin, no diazo reaction. Widal reaction positive.

For the next three weeks the patient ran a course of mild typhoid fever without complications; the temperature rarely rose above 102.5°, and for a large portion of the time was below 102°. The patient suffered no abdominal pain, and there were no intestinal hemorrhages.

On February 14th, the thirty-first day of the disease, the temperature began to rise, and the patient suffered a well-marked relapse. During the next twelve days the symptoms were somewhat more aggravated than during the primary attack; the temperature ran somewhat higher, though at no time was extreme; on several occasions there was nausea and a limited degree of vomiting. There was no abdominal pain, the abdomen was soft, not distended. Urine contained a faint trace of albumin; diazo reaction positive. Patient was somewhat constipated; there were no intestinal hemorrhages.

On February 26th, the forty-third day of the disease and the twelfth day of the relapse, the patient was given an enema at 9 A.M., which was but slightly effectual. Following this he complained of feeling a little distended, and there was slight abdominal pain. This pain, however, soon passed off, and at 10 A.M. the patient was found sleeping quietly; pulse 100. When seen fifteen minutes later he was in a condition of evident shock; he was cyanotic and trembling; the pulse had reached 120, and was very compressible; temperature unaffected. There was quite severe general abdominal pain, the abdomen was somewhat distended, but not very sensitive. The liver appeared to be pushed upward. The patient vomited once, about an ounce of greenish

fluid. Leucocytes 31,600 per cubic millimetre. The patient was seen with Dr. D. P. Allen at 10.30 A.M. Cyanosis was still present. Pulse was 130, low tension. Respirations were 30, thoracic. The abdomen was moderately distended; there was general, not localized, sensitiveness, and there was marked muscular rigidity on the left side; peristalsis was present. The heart sounds were weak, the lungs clear. Patient's condition was fairly favorable for operation.

Operation, February 26th, 11 A.M., by Dr. D. P. Allen, one hour after appearance of symptoms. Local anæsthesia, cocaine solution, 1 per cent., preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty-five minutes.

Incision six centimetres long in right linea semilunaris opposite anterior superior spine, carried into peritoneal cavity without causing the patient pain. Abdominal cavity contained a little brownish fluid. Cæcum readily found, normal. Appendix normal. Perforation of ileum about forty-five centimetres from cæcum, on free border, about two millimetres in diameter. Perforation closed without excision by two rows of continuous silk Lembert sutures. No other perforations or threatening perforations found, but the ileum was not examined beyond the lesion. Abdomen irrigated with large quantities of hot sterile water with flush tube, intestines not being removed; special attention was given to the lumbar fossæ and pelvic cavity. Ileum considerably congested. Peritoneal surfaces otherwise apparently unaffected. No fibrinous flakes. No adhesions seen. Glass-tube and gauze drainage placed to seat of suture, on either side of which the wound was closed with interrupted silkworm-gut sutures. The patient's condition did not appear to be affected by the operation; the pulse was as good as before operation. Very little pain was experienced except during irrigation, when it was moderate.

Subsequent treatment of the case was by strychnine, nutrient and stimulating enemata, subcutaneous saline infusions, sponge baths, and liquids by mouth. The patient appeared to be unaffected by the operation. During the next two days the course was that of an increasingly severe typhoidal toxic condition with the accompanying evidence of exhaustion during the second day. There was failing pulse, marked trembling, moderate delirium at times. The abdomen was only slightly distended, was generally tympanitic, and was but moderately sensitive. At times there was sharp abdominal pain. There was but little discharge from the wound. Urine showed a large trace of albumin, and contained an abundance of hyaline and granular casts. The patient died at 9.10 P.M., February 28th, fifty-eight hours after operation.

Bacteriological examination of the free abdominal fluid at the time of operation showed on cover-slips a few cocci and numerous bacilli. Tube cultures showed a pure growth of staphylococcus pyogenes aureus.

Autopsy, restricted. The intestinal sutures were intact, the perforation being firmly closed. No additional perforations found. There was no general peritonitis. There were no peritoneal adhesions except those about the gauze packing. Cultures and cover-slip preparations from general peritoneal cavity were negative.

CASE IV.—Female, aged sixteen years; very severe case. At entrance leucocytes 9932 per cubic millimetre; Widal reaction positive; diazo reaction positive. Bowels moved readily with enema, no hemorrhages. Marked delirium. Symptoms of perforation on the

fifth day of the disease; abdominal pain, rigidity, distention; rise in pulse and temperature; no vomiting; leucocytes 15,080 per cubic millimetre; general constitutional change marked. Exploration advised.

Operation. About four hours after first appearance of symptoms. Cocaine solution ½ per cent. Time, twenty minutes. Short right lateral incision. No perforation; peritoneal cavity apparently normal. Wound sealed with collodion dressing. Cover-slips and cultures from abdomen negative. Medical treatment uninterrupted by operation. Subsequent course of fever severe; convalescence slow.

H. B., medical No. 1035. Service of Dr. H. S. Upson. Female, aged sixteen years, colored, American, single, cleaner. Admitted January 4, 1900; discharged March 24th. Cured.

Family History. Negative.

Previous History. Children's disease; no other illness recalled; habits good.

Present Illness. Patient was transferred from gynecological service complaining of general weakness. Two days before patient complained of general malaise and ached all over. There had been a little cough, occasional epistaxis, anorexia, and vomiting with moderate frequency. Bowels regular.

Physical Examination. Patient well nourished and developed; mind clear; tongue coated. Pulse 100, fair strength and volume, slightly dicrotic. Temperature, 104.2°. Lungs and heart negative. No enlargement of spleen perceptible. Abdomen soft, no rose-spots. Leucocytes, 9932 per cubic millimetre; hæmoglobin, 85 per cent. Urine, albumin faint trace; diazo reaction present. Widal reaction positive.

From the start the symptoms were those of a very severe typhoidal infection. The temperature was high, averaging 104° to 105°. The reaction from the tub baths was not satisfactory, and the drops in temperature very moderate. The pulse for the first three days averaged 110 to 120, but was of fair quality. There were marked nervous symptoms, the patient being delirious most of the time and rarely giving an intelligent answer to questions. The amount of stimulation required was considerable.

On January 6th the patient complained somewhat of abdominal pain, and there was slight distention but no rigidity. On January 7th, the fifth day of the disease, the patient's condition began to get markedly worse in the early part of the evening. The patient was delirious, so that her subjective sensations were of little avail in diagnosis. In spite of the delirium, however, she complained of abdominal pain, which it seemed must be at least moderately severe to arrest her attention at all. The abdomen was moderately distended, and was everywhere tympanitic. There was a considerable degree of muscular rigidity. No definite sensitiveness could be localized. The pulse had risen from 120 to 140, was weak and dicrotic. The temperature had risen from 102.8° at 5 P.M. to 105.5° at 8 P.M. The leucocytes at 8 P.M. were 15,080. The patient was seen about 8 P.M. with Dr. G. W. Moorehouse, and essentially the same condition found as noted above. A definite diagnosis of perforation could not be made. The constitutional effect, however, from some cause or other had been very marked, and owing to the delirious condition of the patient and apparent insensibility to ordinary degrees of pain it was felt that a perforation under

the circumstances would be very easily overlooked, and that the physical condition was such as one might readily expect in a patient so severely ill if a perforation had actually occurred. On account, therefore, of the possibility of overlooking a perforation, together with the degree of abdominal symptoms afforded by the patient in even such a delirious condition, an exploration under local anæsthesia was advised. The risk of such an operation was very slight, whereas the danger from an unrecognized perforation was of the gravest possible character.

Operation, January 7th, 9.45 P.M., by Dr. C. E. Briggs, about four hours after the appearance of symptoms. Local anæsthesia, cocaine solution, ½ per cent., preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty minutes.

Incision three centimetres long in right linea semilunaris opposite anterior superior spine. On opening the peritoneum no free fluid was found. There was no injection of the cæcum or adjoining portions of the ileum. No adhesions were found, and the peritoneal cavity appeared to be perfectly normal. Cover-slip preparations from right iliac fossa and pelvis showed no organisms. Wound closed with buried silk sutures in layers. A collodion dressing was applied, consisting of several alternate thin layers of sheet wadding and collodion, making a firm and absolutely impervious dressing. Condition of patient apparently unaffected by operation. Permission given to continue baths within four hours if desirable. Patient remained on the medical service.

The subsequent history of the case is of little interest from the present stand-point, except for one attack of abdominal pain. She ran the course of a very severe typhoidal infection, the toxic symptoms being so severe on several occasions that her recovery seemed hopeless. The temperature remained persistently high for nearly ten days, and was but slightly affected by tub baths. On only one occasion, January 20th, the eighteenth day of the disease, was there any subsequent suggestion of an abdominal complication. On this day she was awakened at 2 A.M. by severe abdominal pain. The pulse was but slightly affected. The temperature was unchanged. The abdomen was soft and not sensitive, and there was very slight sensitiveness on deep pressure in right lower quadrant. The pain could not be definitely localized, although the patient was perfectly rational, and it passed off within a short time. The leucocytes, however, at 2.30 A.M. had risen to 50,660 per cubic millimetre; two days before the leucocytes were 8060, and five days later 9600 per cubic millimetre. The convalescence was slow and uneventful. Following the operation, baths were given as required without reference to the incision. The incision healed by first intention. The patient was discharged March 24th, cured.

Bacteriological Examination. Cultures taken from the right iliac fossa and pelvis showed no organisms in cover-slip preparations, and there was no growth on cultures.

CASE V.—Male, aged seventeen years; mild case. At entrance leucocytes 4900 per cubic millimetre; Widal reaction positive; diazo reaction positive. Bowels rather constipated, no hemorrhages. Symptoms of perforation on twentieth day of disease; severe abdominal pain, general sensitiveness, slight distention, marked muscular rigidity in lower half of right side; rise in pulse; fall in temperature of 1°; no vomiting; leucocytes 9200, 13,000, 13,200 per cubic millimetre. Condition of patient favorable for operation.

Operation. Three hours after first appearance of symptoms. Cocaine solution, 1 per cent.; chloroform. Time, fifty-five minutes—forty minutes under chloroform. Right lateral incision. Yellowish-gray abdominal fluid. Perforation eleven centimetres from cæcum, closed with continuous Lembert silk sutures; no threatening perforations found. Irrigation with sterile water. Intestines not removed. Glass-tube drainage from pelvis. Cultures showed bacillus mucosus capsulatus. Recovery.

C. W., medical No. 1208; surgical No. 1530. Service of Dr. E. F. Cushing. Male, aged seventeen years, white, American, single, laborer. Admitted June 16, 1900; discharged August 19th. Cured.

Family History. Negative.

Previous History. Has always been well and strong. A few years ago had erysipelas, and later a fever the nature of which he did not know. Habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, backache, and pain in stomach. Two weeks previously symptoms began with general aching sensation and tired feeling. Shortly after this he began to have nausea, and there was anorexia. Epistaxis was frequent. Bowels rather costive. Had been in bed one week.

Physical Examination. Fairly well nourished and developed; mind clear; face flushed; lips dry; tongue coated. Pulse 80, full, but readily compressible, dicrotic. Temperature 101.8°. Lungs and heart negative. Spleen apparently not enlarged. Abdomen not distended, soft, and showing numerous rose-spots. Leucocytes 4900 per cubic millimetre; hæmoglobin, 85 per cent. Urine, albumin faint trace; diazo reaction present. Widal reaction positive.

During the next five days the symptoms were those of a rather mild case of typhoid fever, the temperature coming down gradually, with good drops following tub baths. There was no diarrhœa, no intestinal hemorrhages. The abdomen was soft and flat. The progress of the case was very satisfactory.

On June 22d, the twentieth day of the disease, the patient had been as usual all through the day. No tub baths had been given. At 6.30 P.M. he was seized with a sharp abdominal pain which was not definitely localized. When seen a few minutes after this there was no abdominal distention, but marked muscular rigidity and general sensitiveness. The pulse was 76 and of good quality; the temperature 102.8°, which was 1° higher than at 4 P.M. The leucocytes at 6.45 P.M. were 9200 per cubic millimetre. The pain was sufficient to make the patient groan audibly. A small enema with turpentine, two drachms, was given, which was followed by a light-yellow stool and a small amount of gas, but afforded no relief from the pain. Morph. sulph., gr. one-quarter, was given hypodermically. At 7 P.M. the pulse was 88, and the temperature had fallen 1°. At 7.30 P.M. the patient was seen with Dr. J. L. Martin. The pulse was 110, low tension, readily compressible. The patient had a dusky appearance, and there was marked cyanosis of the lips and finger-nails. Respirations were shallow, mostly thoracic. The abdomen was not distended, but there was marked rigidity over the lower half, and was a little more evident on the right side; there was considerable general sensitiveness. The patient still complained of moderately severe pain in lower half of abdomen. At 8 P.M. the pulse had risen to 120. The cyanosis was slightly less marked,

but otherwise the condition was about the same. The leucocytes were 13,000 per cubic millimetre. At 8.30 P.M. the leucocytes were 13,200 per cubic millimetre.

Operation, June 22d, 9.30 P.M., by Dr. C. E. Briggs, three hours after first appearance of symptoms. Local anæsthesia, cocaine solution, 1 per cent., preceded by morph. sulph., gr. one-quarter, hypodermically; chloroform. Time, fifty-five minutes, forty minutes of which were under chloroform.

On reaching the operating-table the abdominal pain was still present, and there was slight distention. The cyanosis was considerably less. Pulse 120. Under local anæsthesia an incision was made six centimetres long in right linea semilunaris. Although the patient suffered no pain, he became very nervous and quite beyond control, and light chloroform anæsthesia was substituted. Owing to the boy's nervous condition the opening of the abdomen under cocaine required great patience, consuming fifteen minutes. On opening the peritoneum a moderate amount of thin, yellowish-gray fluid escaped. Cæcum readily found, ileum reached, and perforation found on its free border about eleven centimetres from the ileocæcal valve. It was about two millimetres in diameter and exuded bowel contents when pressed. About it the bowel was red and thickened for about two centimetres in diameter. The perforation was wiped off with bichloride and turned in with two rows of running fine silk Lembert sutures for a length of three centimetres. Over these were placed three interrupted Lembert sutures to relieve the strain, as the bowel was very friable. The cæcum and ileum for about one metre were searched with eye and finger for perforations or thin ulcers, but nothing suggesting a threatening perforation was found. The ileum for about half this distance was injected and seemed somewhat thickened. Appendix normal. No fibrinous flakes were observed. No adhesions were found. Incision enlarged to nine centimetres. Abdomen was thoroughly irrigated with a large amount of sterile water, with special reference to the pelvis and flanks; the intestines were not removed. The pelvis contained a large amount of thin, purulent-looking material, rather different in appearance from that seen on opening the abdomen. Examination of cover-slip preparations from this fluid showed numerous large and small bacilli and a few small cocci. Intestinal coils near pelvis were injected, but remainder of abdomen appeared nearly normal. Abdomen flushed till perfectly clear. Glass drainage-tube placed in pelvis, brought out through lower end of wound, above which the wound was closed with interrupted silkworm-gut sutures. The site of perforation was left immediately below the incision. During the operation the patient's pulse remained at 120, good quality, rising a little during recovery from anæsthesia. Patient was but very slightly under chloroform, and was moving most of the time except during closure of the abdominal wall. The forty minutes under chloroform were largely spent in careful examination of the bowel, and in very thorough irrigation of the peritoneal cavity.

The subsequent treatment of the case was by nutrient and stimulating enemata, strychnine, sponge baths, and liquids by mouth. During the first night there was very profuse sweating, which was relieved by atropine. The day after the operation, June 23d, the patient began to vomit a dark-brown fluid, and this continued for several hours, the patient's general condition becoming considerably affected. The

stomach was washed out, about three pints of dark-greenish fluid being evacuated, after which the vomiting ceased, and there was immediate and steady improvement.

From this time on the patient made a progressive and uninterrupted recovery, the pulse coming down to between 80 and 90 on the second day after the operation, and the temperature falling gradually to 99° by the end of the first week. The leucocytes on the second, sixth, and eighth days after operation were 15,400, 5400, 5600 per cubic millimetre, respectively. The discharge from the wound was moderate. The glass tube was gradually withdrawn, being replaced by gauze packing on the eighth day, which was entirely omitted four days later. The wound was very indolent, and there was considerable sloughing, but no active suppuration. The wound was entirely healed four weeks after operation, but owing to the persistence of a slight degree of fever —99° to 99.5°—the patient was kept in bed nearly two weeks longer. The cause of this temperature was never discovered. The patient was discharged August 19th, fifty-eight days after operation, in excellent condition.

Bacteriological Examination. Cover-slips and cultures taken from the abdominal fluid when the peritoneum was first opened were negative. Cover-slips taken from the seat of the ulcer and from the turbid pelvic fluid mentioned showed medium-sized bacilli and a few small cocci. Plate cultures from these two localities showed bacillus mucosus capsulatus. Cultures taken from the bottom of the drainage-tube four days after operation gave a pure growth of the same organism.

CASE VI.—Male, aged twenty-eight years. Patient entered hospital with general peritonitis; no history obtainable. At entrance leucocytes 10,600 per cubic millimetre; Widal reaction positive; diazo reaction positive. Diarrhœa followed by constipation; no intestinal hemorrhages. Marked abdominal distention, rigidity, pain, and sensitiveness. Date of perforative symptoms not determined.

Operation. Period after perforation not known. Ether. Time, thirty minutes. Right lateral incision. Turbid abdominal fluid. Perforation of ileum thirty centimetres from cæcum; closed by continuous Lembert catgut sutures. Irrigation with sterile water; intestines not removed. Gauze and glass-tube drainage. Cultures from abdominal fluid, unidentified bacillus, probably bacillus coli communis, or bacillus mucosus capsulatus. Death four hours after operation, general peritonitis.

Autopsy. Intestinal sutures intact, no other perforations. General peritonitis. Cultures from heart, lungs, kidneys, and brain, bacillus mucosus capsulatus; from general peritoneal cavity, negative.

J. K., medical No. 1302; surgical No. 1660. Service of Dr. E. F. Cushing. Male, aged twenty-eight years, white, German, single, laborer. Admitted August 20, 1900; discharged August 21st. Dead.

History was obtained subsequently for purposes of record, but no information was at hand at time of entrance for aid in diagnosis.

Family History. Negative.

Previous History. No former illnesses known. Habits good.

Present Illness. Patient entered hospital complaining of abdominal pain. The symptoms began with headache eight days previously. For the last six days he had abdominal pain with some distention. There was diarrhœa at first, but during the last few days there was but one

movement. There was no vomiting or epistaxis. No fever was noted. Was in bed only part of this time. Abdominal pain had been constant.

Physical Examination. Patient entered in the evening, and no history was available to aid in diagnosis. Moderately well developed, poorly nourished. He had the appearance of a very sick man. Tongue coated and dry. Pulse 120, irregular, but moderately strong. Temperature 100.8°. Lungs: on right side below clavicle there was slightly increased vocal fremitus; otherwise negative. Heart appeared not enlarged, sounds weak. Liver dulness from fourth to sixth rib in mammary line. Spleen: dulness anterior to about mid-axillary line; palpation impossible on account of distention. Abdomen distended, tympanitic, moderately rigid, sensitive to pressure, more marked in lower half and possibly slightly increased on right side; no rose-spots; doubtful dulness in both flanks. Leucocytes 10,600 per cubic millimetre at 9 P.M. Urine: albumin ½ per cent.; diazo reaction faint. Widal reaction positive; both of these examinations made the following morning.

The case was thought to be one of general peritonitis from perforation of a typhoid ulcer, although the existence of typhoid fever was not absolutely determined until the next morning. Circumstances, however, made an immediate operation impossible. During the night he was very actively stimulated. The next morning the Widal reaction determined the diagnosis. The case was seen by Dr. D. P. Allen about 9 A.M. The abdominal distention was extreme. There was general tympany, marked muscular rigidity, and general sensitiveness. The patient was in a condition of profound shock. Pulse 140 and very poor quality; temperature 105.6°; respirations 40. The case was considered practically hopeless.

Operation, August 21st, 10.30 A.M., by Dr. D. P. Allen. Time, after onset of symptoms not known. Anæsthetic, ether. Time, thirty minutes.

Incision seven centimetres long in right linea semilunaris opposite anterior superior spine. On opening the peritoneum a moderate quantity of straw-colored fluid and thin pus escaped, containing fecal matter. Perforation of the ileum was found on the free border about two millimetres in diameter, about thirty centimetres from the cæcum. This was closed with two rows of running catgut sutures, strengthened by four interrupted silk sutures. There was considerable agglutination of the intestinal coils in the neighborhood of the perforation, together with congestion of the remaining coils of intestines and the parietal peritoneum. No other perforations found. A considerable amount of thin pus was found high up under the liver and a considerable amount in the pelvis. The abdomen was thoroughly irrigated with sterile water, the intestines not being removed. Gauze and glass-tube drainage from pelvic cavity and subhepatic region. Pulse during the operation 180, very weak, often imperceptible. The patient was highly stimulated with strychnine and subcutaneous saline infusions. There was no reaction, however, and he died at 3 P.M., four hours after the operation.

Bacteriological Examination. Cover-slips from the abdominal fluid showed cocci and small bacilli. Tube cultures showed a pure culture of bacillus coli communis or bacillus mucosus capsulatus, the exact identity apparently not having been established.

Autopsy, complete, No. 194, August 26th. Anatomical diagnosis, typhoid fever, ulceration, and perforation with general peritonitis; apical tuberculosis and hypostatic pneumonia; acute splenic tumor. Special lesion; thirty centimetres above cæcum there was a perforating ulcer of the ileum, closed with catgut sutures, which were still intact; there were several other ulcers with very thin bases in the neighborhood, but no perforations. Cultures from heart, lungs, kidneys, and brain showed bacillus mucosus capsulatus; from general peritoneal cavity, negative, owing probably to the recent irrigation.

General Considerations. That all six cases were typhoid fever is readily established. The Widal reaction was positive in four cases —III., IV., V., and VI. In the first two cases of the series the Widal reaction was negative at entrance, and for some reason was not recorded later. The course of the disease was perfectly typical in all instances. A perforation was found in all cases except Case IV., and this case had a positive Widal reaction. Autopsy in the fatal cases—I., II., III., and VI.—showed typical bowel lesions.

The cases with one exception—Case V.—were all severe infections, and this severity, not the complication, was held responsible for the death in two cases—I. and III. One case—III.—occurred during a moderately severe relapse, following a rather mild primary course.

The bowels were constipated in three cases—II., III., and V.—in the first of which the condition was very marked. In one case—Case I.—there was moderate diarrhœa. In Case IV. the bowels moved easily with enema, neither marked constipation nor diarrhœa being observed. In Case VI. the bowels were loose at first, followed by rather aggravated constipation.

Intestinal hemorrhages occurred in only one instance—Case II. In this case it was present on the seventeenth and eighteenth days of the disease, was slight in amount, but was accompanied with considerable abdominal pain. The last hemorrhage was apparently coincident with the perforation.

The ages were sixteen, seventeen, seventeen, twenty-eight, thirty-one, and forty-seven. There was but one female in the series—Case IV. She was colored, and no perforation was found. The remaining cases were males, and white. In Finney's series but one case occurred in a negro, and that was fatal.

Signs and Symptoms. As compared with the large proportion of the more extended collections, the cases presented show a remarkably distinct set of symptoms. In Case VI. the patient entered with unmistakable signs of general peritonitis, and the primary symptoms of perforation in this case were not observed. The discussion of the signs and symptoms is restricted, therefore, to the first five cases.

The date of appearance of symptoms was the fifth day in Case IV.; the eighteenth day in Case II.; the twentieth day in Case V.; the

twenty-first day in Case I.; the forty-third day, the twelfth day of a relapse, in Case III. In Case VI., judging from the probably inaccurate history, the perforation occurred early, probably on the fifth or sixth day.

The abdominal symptoms were quite well marked in all cases. Pain appeared suddenly in three cases—Cases II., III., and V., and came on gradually in two cases—Cases I. and IV. It was confined to the lower half of the abdomen in three cases—Cases I., II., and V., but in Case I. it eventually radiated to the upper half and became general. In Case III. it was general, and in Case IV. it appeared to be general, but the delirious condition of the patient made accurate observation impossible. The pain was severe in three cases—Cases II., III., and V.—and moderate in two cases—Cases I. and IV. In Case I. pain also radiated to the penis, which was the only instance. Sensitiveness to pressure was slight in two cases—Cases II. and IV.; moderate in Case III.; marked in Cases I. and V. It was general in three cases—Cases III., IV., and V., and was confined to the lower half of the abdomen in two cases—Cases I. and II. Increased sensitiveness in the right lower quadrant so frequently spoken of was not observed.

Muscular resistance was a very marked sign in all but one of the cases—Case IV. In this instance the resistance was but moderate, and no perforation was found. The resistance was general in three cases—Cases I., II., and IV., and was confined to the right half of the abdomen in one case—Case III. In Case V. it was confined to the lower half of the abdomen, and was considerably more marked on the right side. Distention was observed as moderate in Cases I., II., III., and IV., but was present to a slight degree only in Case V. Tympany to a greater or less extent was observed in all of the cases. Slight vomiting was observed in Cases I., II., and III., and was absent in Cases IV. and V.

The pulse showed a marked rise in all instances—20 to 50 beats. The quality of the pulse also became progressively worse with the increase in the number of beats, and was a very valuable sign. In Case IV., where no perforation was found, the pulse rose very gradually from 120 to 140. The marked severity of the case, however, made all of the signs and symptoms in this instance particularly difficult to estimate with much accuracy. The temperature was unaffected in two cases—Cases II. and III.—and in two cases—Cases I. and IV.—there was a distinct rise, 101° to 102.2°, and 102.8° to 105.5°, respectively. In but one case—Case V.—was there a fall in temperature, and in this instance of but 1° only, 102.8° to 101.8°. The respiratory rate was markedly increased. The nature of the respirations, however, was noted as largely thoracic in all the cases. This was especially marked, together with a very shallow quality, in Case V.

The condition of general systemic shock, as observed shortly before operation, was very marked in Case II., and was present to a considerable degree in Case IV. It was noted as moderate in Cases I., III., and V. Cyanosis, especially of the lips and finger-nails, was marked in Case V., moderate in Case III., slight in Cases I. and II., and absent in Case IV.

The white blood count as recorded showed a distinct rise in all but one instance—Case I. The records are as follows: Case I., entrance, 5700 per cubic millimetre; perforation, six days later, 4460 per cubic millimetre. Case II., entrance, 5980; perforation, eight days later, no count recorded. Case III., entrance, 5700; perforation, thirty-two days later, 31,600. Case IV., entrance, 9932; symptoms of perforation, two days later, 15,080. Case V., entrance, 4900; perforation, five days later, 9200, 13,000, 13,200. Case VI., entrance, 10,600. It is to be noted in Case IV. that subsequent to the operation the leucocytes on one occasion rose from 8060 to 50,660, associated with considerable abdominal pain, but no other constitutional effect.

The Condition of Patient at Operation. The cases with but one exception—Case V.—were rather unfavorable to operation, two being quite so, and one practically hopeless. As this is regarded as of considerable importance, the conditions will be given in more detail: Case I. The patient's condition was poor, although not absolutely unfavorable to operation. The perforation occurred on the twenty-first day of the disease, in a moderately severe case, before the patient had begun to recover at all from the effects of his illness. He was weak and emaciated, and had comparatively little recuperative power. Case II. This case was quite unfavorable for operation. The patient was a strong, athletic boy, but the case had run a severe course, and the perforation occurred on the eighteenth day during the very height of the disease. The patient was weak and exhausted, and the rapidity with which he succumbed to the peritoneal infection was not surprising. Case III. This case was somewhat more favorable, although not very promising. The perforation occurred during the height of a rather severe relapse, being the forty-third day of the disease, the twelfth day of the relapse. He was quite weak, but appeared at the time to have a fair degree of strength. Case IV. The case was desperately sick at the time perforation was feared, but the symptoms appeared so early, the fifth day, that the patient's constitution had not been badly reduced. On this account it is believed she would have stood fairly well a more extensive operation. Case V. The condition of this patient was very favorable for operation, in marked contrast to the other cases. The case was mild, and when the perforation occurred, the twentieth day, the temperature had begun to come down; he was not badly reduced. Case VI. The case, as stated, was *in extremis* at entrance, and was regarded as practically hopeless.

Duration of Symptoms. The lapse of time between the first appearance of symptoms and the operation was short in all but one instance—Case II. In Case III. the operation was performed one hour after the appearance of symptoms. Cases V., IV., and I. were operated upon within three, four, and five hours, respectively, although the exact time of suspected perforation in Case IV. was somewhat indefinite, owing to the delirium of the patient. Case II. was opened eight hours after definite symptoms first appeared; three days previously the patient had had abdominal pain following a tub bath, which grew less the next day, disappeared entirely the day before active symptoms, and was entirely unassociated with constitutional signs suggesting perforation. The duration of symptoms in Case VI. could be only roughly estimated, as perforation occurred before entrance, but it was probably twenty-four to forty-eight hours.

Operation. The technique of the operations presented very little that was distinctive. The anæsthetic was general in four cases, local in two cases. Chloroform, very lightly given and preceded by morph. sulph., gr. one-quarter, was administered in Cases I., II., and V. Ether was similarly given in Case VI. Local anæsthesia, cocaine solution, ½ and 1 per cent., preceded by morph. sulph., gr. one-quarter, was employed in Cases III. and IV. In Case V. the abdomen was entered under cocaine, but the nervous condition of the patient required the administration of chloroform.

The incision in each instance was in the right linea semilunaris opposite the anterior superior spine, three to nine centimetres long. In each instance, except Case IV., the cæcum was readily found, the ileum, cæcum, and appendix examined. In Case IV. the peritoneal cavity was so visibly normal that the ileum was not withdrawn. This, however, was considered a fault in the operation. An examination of the ileum was made for about one metre in Cases I., II., and V., but in Cases III. and VI. the inspection was not carried beyond the perforation. In all cases careful search was made for thin areas, threatening perforations, as well as for complete perforations. The perforation was excised in no instance. The closure of the perforation was by two rows of continuous Lembert sutures in each case, in two of which—Cases V. and VI.—these were strengthened by another line of interrupted silk sutures. The suture material was catgut in Cases I. and VI., and silk in the remainder. The irrigation was with very large quantities of sterile water, by means of a large flushing tube. With this tube all parts of the peritoneal cavity were readily reached, special attention being given to the lumbar fossæ and pelvic cavity. Only the portion of the ileum examined was removed from the abdomen, and an effort was made to handle the intestines as little as possible. Drainage was used in all cases except the exploration—Case IV. Iodoform gauze

alone was used in Cases I. and II.; glass-tube and iodoform gauze in Cases III. and VI., and a single glass drainage tube from the pelvis in Case V. The site of the suture was left close to the abdominal wall in all instances. The wounds were all partially closed with interrupted silkworm-gut sutures through all layers, except Case IV., which was completely closed with interrupted buried silk sutures. The time of operation was twenty minutes in one case, twenty-five minutes in two cases, and thirty minutes in two cases. In Case V. fifty-five minutes were consumed, fifteen minutes in going through the abdominal wall under cocaine, and forty minutes, or the remainder of the operation, under chloroform. The condition of the patient was apparently unaffected by the operation in Cases III. and IV.; it was excellent in Case V.; was poor in Cases I. and II., and very bad in Case VI.

PATHOLOGY. Pathological observations of the cases are of special interest, and it is to be greatly regretted that more complete autopsies could not be obtained.

There was a moderate degree of peritonitis at the time of operation in all the cases of perforation, judging from the amount of free fluid in the abdominal cavity. In Case I. there was considerable dark-brown fluid with fecal odor; in Case II. a moderate amount of dark fluid; in Case III. a small amount of brownish fluid; in Case V. a moderate amount of thin, yellowish-gray fluid, and in the pelvis a large amount of thin, purulent-looking fluid; in Case VI. a moderate amount of straw-colored fluid with a large accumulation of thin, purulent fluid under the liver and in the pelvis.

The perforation was single in each instance, and occurred on the free border of the ileum. In only one case—Case II.—was a threatening perforation found. The perforations were all small, two to three millimetres in diameter, and were surrounded by an indurated friable base about two centimetres in diameter in several instances. In one case—Case II.—there was a small amount of fibrinous exudate in the neighborhood of the perforation. The perforations were eight, eleven, twenty-five, thirty, and forty-five centimetres from the cæcum in Cases I., V., II., VI., and III., respectively.

The ileum, aside from the perforation, was altered in each instance, ranging from slight congestion to a thickened dark appearance. The appendix was normal in all cases. The cæcum was appreciably congested in Case I. only. The remaining coils of intestines were slightly congested in Case I. In Case V. those lying in the pelvis were considerably congested; in Case VI. there was a marked congestion, and agglutination of the coils in some places. The parietal peritoneum was appreciably congested in Cases I. and VI. only. In none of the cases were there fibrinous flakes in the peritoneal cavity, except for a small amount of deposit about the perforation in Case II. No adhesions

were observed in any of the cases except in Case VI. In Case IV., in which the symptoms appeared on the fifth day and no perforation was found, the abdominal cavity was perfectly normal so far as could be observed.

Bacteriology. Bacteriological examination of the free abdominal fluid was made in all cases, but in only one instance was a culture taken from the perforation. Cover-slip preparations from Case I. showed a medium-sized bacillus and a small coccus; plate cultures grew bacillus coli communis only. Case II. showed a pure growth of bacillus coli communis in plate cultures. Case III. showed in cover-slips a few cocci and numerous bacilli; in plate cultures, a pure growth of staphylococcus pyogenes aureus. Case IV., in which no perforation was found, cover-slips and cultures were negative. Case V., cover-slips and cultures from fluid on first opening the abdomen were negative; cover-slip preparations from the perforation and the turbid pelvic fluid showed numerous medium-sized bacilli and a few cocci. A plate culture from these two localities gave a pure growth of bacillus mucosus capsulatus; a tube culture taken from the bottom of the drainage-tube in the pelvis four days after operation showed a pure culture of the same organism. In Case VI. cover-slip preparations showed cocci and small bacilli, the nature of which was not definitely determined, but was apparently bacillus coli communis or bacillus mucosus capsulatus.

Autopsies, complete, Case VI., or restricted, Cases I., II., and III., were made in the four cases of death. General peritonitis was present in two cases only—Cases II. and VI.—in the other two cases the general peritoneal cavity being free from fluid and adhesions, and the cultures from the same being negative. Case I., death seventy-five hours after operation, showed a local peritonitis about the gauze packing, into which the original perforation and four small neighboring perforations opened; the catgut sutures were almost entirely absorbed; no general peritonitis; the ileum showed several large confluent ulcers, not perforating. Cultures from the local peritonitis showed bacillus coli communis; cover-slips and cultures from general peritoneal cavity were negative. Case II., death sixteen hours after operation, sutures intact, no other perforations; general peritonitis, cultures growing bacillus coli communis. Case III., death fifty-eight hours after operation; intestinal sutures intact, no additional perforation; no general peritonitis, cover-slips and cultures from general peritoneal cavity negative. Case VI., death four hours after operation; suture intact, no other perforations: general peritonitis; cultures from heart, lungs, kidneys, and brain showed bacillus mucosus capsulatus; cultures from general peritoneal cavity were negative, probably owing to recent irrigation.

Results. In considering the results of these cases from the standpoint of operations for the relief of typhoid perforation, it is manifestly

improper to include Case VI., which was suffering from general peritonitis before the case was seen at all, and was operated upon solely for the relief of the abdominal infection. Case IV., in which no perforation was found, adds one other to the increasing list of explorations without untoward results in cases of suspected perforation, but cannot, of course, be included in the list of cases where perforation has actually occurred. In the four remaining cases with perforation, one alone recovered, Case V. In all four cases there was a sufficient degree of peritonitis to cause an accumulation to a greater or less degree of free abdominal fluid, in which organisms were found in both cover-slip preparations and cultures. Only one case—Case II.—as shown at autopsy, died of general peritonitis. In this instance the operation was deferred eight hours, the longest delay in the series, and an evident mistake. The other two fatal cases—Cases I. and III.—showed local peritonitis about the packing, as occurs in any abdominal wound that is drained, but the general peritoneal cavity was free from fluid and adhesions, and no organisms were obtained from it in cover-slip preparations or cultures. It can be safely said, then, that whatever else one may wish to consider the cause of death in these two cases, it was not general peritonitis. A very large percentage of the fatal cases following operation so far reported, at least 80 per cent., died from general peritonitis. The percentage cannot be given with any great degree of accuracy, however, owing to the small number of post-mortem examinations. The fact that it was possible to prevent the occurrence of general peritonitis in three out of four cases of perforation is worthy of note despite the fact that only one case recovered. The two fatal cases without general peritonitis—Cases I. and III.—lived seventy-six and fifty-eight hours, respectively, after operation. In Case III. the perforation occurred at the height of a moderately severe relapse, in which the clinical evidence of increasing typhoidal toxæmia was apparent before operation. The operation was performed under local anæsthesia without shock to the patient that was apparent to careful observation. It is confidently believed that this death was due to typhoidal toxæmia, and was independent of the operation. As a complete autopsy was not obtainable, however, this statement cannot be maintained with absolute certainty, although it is very strongly supported by clinical symptoms and the absence of general peritonitis. In Case I. perforation occurred on the twenty-first day during the height of the fever in a moderately severe case, before the patient had begun to recover from the effects of the disease, and it was evident that he had very little recuperative power. The clinical appearance of the patient on the last day of life was one of exhaustion and gradual heart failure, and was not unexpected, owing to the condition of low vitality which was apparent before operation. This of course was

added to very materially by the shock of operation, which was, unfortunately it is believed in this instance, performed under general rather than local anæsthesia. It was also undoubtedly added to in a considerable degree by four subsequent perforations near the original lesion. The closure of the perforation was not successful, owing to the subsequent absorption of the catgut sutures. By rare good fortune the four subsequent perforations had opened along the line of drainage, and the extravasation was being cared for completely, as far as could be seen at autopsy, by the gauze drainage going down to the original perforation. The general peritoneal cavity was entirely free from evidences of peritonitis, there being no fluid or adhesions, and the cover-slip preparations and cultures from the same being negative. It is believed that the patient died of exhaustion. It is certain he did not die of general peritonitis. Here, again, however, the absence of complete autopsy leaves the absolute cause of death somewhat in doubt.

TWO CASES OF TYPHOID FEVER COMPLICATED BY NOMA.

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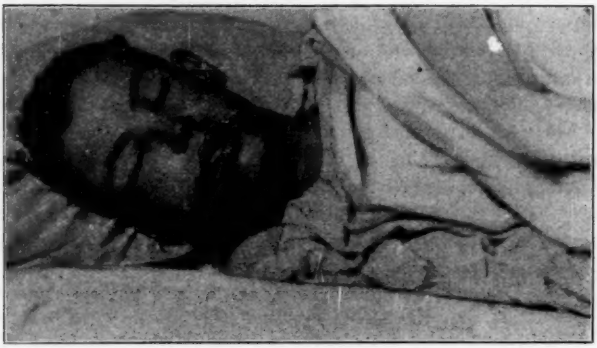
(From the Service of Professor John H. Musser.)

The following cases are reported partly because of their extreme rarity, partly because they illustrate a serious but, I believe, under certain circumstances, avoidable complication, and partly because they furnish additional proof of the etiological nature of a hitherto obscure condition.

Case I.—T. T., white, aged fourteen years, a rag-picker by occupation, was admitted to the service of Professor Musser at the University Hospital, July 2, 1901. He had always been a rather delicate boy, and his appetite had never been good. His habits were apparently unexceptionable. His work was very severe, and he was kept at it constantly. His parents were healthy. Four brothers and two sisters were said to be well. Two brothers and one sister have chronic cough with expectoration, but it is not known whether or not this is tuberculous. One brother and one sister died of pneumonia, and the patient stated that it was supposed that his grandmother was suffering from consumption. There was no history of malignant disease in the family. The mother had had one miscarriage. In childhood the patient had measles and spasms. There was no history of influenza or of typhoid fever. For over a year he had had a severe cough with expectoration, and last summer he had an attack of diphtheria. From time to time he had difficulty in speaking, presumably caused by hoarseness. His present illness commenced on June 27, 1901, with headache and pain in the abdomen. He subse-

quently developed fever and diarrhœa, and on July 1 had an attack of epistaxis. When admitted (July 2d) the temperature was 104°, the pulse 112, respiration 48. There was a profuse reddish papulary

FIG. 1.



Fourth day of the noma. July 16th.

FIG. 2.

Sixth day. July 18th.

eruption with white centres over the body; the pharynx was congested, but there was no membrane. The tongue was heavily coated and black on the dorsum. The abdomen was moderately distended, but not painful. The leucocytes were 4320. A diagnosis of typhoid

fever was made and the blood sent to the Pepper Laboratory for the purpose of determining the presence of the Widal reaction. The patient was then sponged and tubbed. The first tub was followed by a fall of 5° in temperature, and the pulse became very weak. From July 4th the Widal reaction was positive. On this day the physical

FIG. 3.

Seventh day. July 19th.

FIG. 4.

Eighth day, showing the improvement after the second injection of antitoxin. July 20th.

examination gave the following results: Heart not enlarged; sounds clear and normal in intensity. Respirations slightly harsh, almost puerile. Liver commenced at sixth rib and extended to the costal border. Respiratory movements were distinct. The spleen was not palpable. The abdomen was slightly distended, and there was some

gurgling in the right iliac fossa. There was an eruption over the body consisting of elevated papules with whitish centres—evidently an acne. Distinct typhoid spots could not be made out. The face was flushed; the tongue broad and black on the dorsum; the lips dry and the seat of a slight herpetic eruption. The throat appeared to be normal. The pulse was medium, full, slow and compressible. The vessels were soft. The knee-jerks were markedly exaggerated. On July 5th the diazo reaction was positive. The urine contained a faint trace of albumin and a few leucocytes. On the following day a distinct and abundant roseolar eruption appeared on the abdomen. The patient's condition continued serious, but not alarming, for several days. From time to time he had an involuntary evacuation of the bowels, and there was some distention of the abdomen. There was cough, with copious dirty brown expectoration, which, when examined, showed numerous forms of micro-organisms, including a form resembling the leptothrix. Tubercle bacilli were not found. On July 12th, the sixteenth day of the disease, it was observed that the right jaw was

FIG. 5.

Ninth day, just before death. July 21st.

swollen and that the right lower molars had become loosened. There was also some ulceration of the mucous membrane of the cheek and of the gum over the right lower alveolar process, and the eruption on the lips was more pronounced. On July 13th three of the lower molars became so loose that they were removed. The patient was seen by Drs. Cryer and Frazier, who confirmed the diagnosis of gangrenous stomatitis. The condition rapidly progressed. The right submaxillary gland became swollen, and a reddish spot appeared on the cheek just below the right corner of the lip. By July 15th a small purplish spot appeared about one-half inch below this, and the ulceration of the interior of the mouth had become very severe and of a dark greenish-purple color. The purple spot on the outside of the face rapidly increased in size in the following manner: First, the area around became reddish, then a blister formed, which was in turn invaded by the gangrene. Dr. Frazier suggested hot poultices of 1 to 8000 bichloride constantly applied to the lesion, and a mouth wash of bichloride. The patient's condition not being satisfactory he was given free stimulation. The

cultures made from the mouth were sent to the Pepper Laboratory and to the pathological laboratory of the medical school for diagnosis. On July 17th the latter reported that the cultures taken from the mouth, which were made only upon agar, showed streptococcus pyogenes and staphylococcus albus. Dr. Kneass, of the Pepper Laboratory, reported that cultures on blood serum gave a typical growth of the Klebs-Loeffler bacillus and other cocci. The patient was immediately isolated and given 4000 units of anti-diphtheritic serum. During the following night the patient rested more easily, the gangrene did not spread, but toward morning he was slightly delirious. The second culture taken from the mouth was examined on July 19th by Dr. Kneass, who again found and demonstrated to me a typical growth of diphtheria bacilli. On July 19th the patient again received 4000 units of antitoxin. By this time signs of perforation had appeared in the gangrenous area. The following day the general condition was much improved, and the local condition had ceased to spread. There was also a very marked decrease in the œdema surrounding the gangrenous area, and the patient was able to open the right eye without difficulty. On July 21st the lesion again commenced to spread and the pulse was weaker. At 2 P.M. 4000 units of antitoxin were given and that night the patient again was slightly delirious. He grew weaker, and finally died at 10 A.M. on July 23d, 1901.

CASE II.—M. T., female, aged eight years, a sister of T. T., was admitted to the service of Professor Griffith on July 8, 1901. The patient had always been delicate, but there was no history of diphtheria or other acute disease. One week before admission she had complained of headache, and had had a slight diarrhœa, poor appetite, and a disposition to sleep most of the time. When admitted she still complained of headache; was slightly delirious; hearing was slightly impaired; the abdomen was not distended or tender; there was slight diarrhœa. Physical examination showed a poorly nourished, pale girl; the tongue was coated and red at the edges; the breath was fetid; the respirations were rapid; the lungs were negative, with the exception of a slight harshness in the right axillary line. The spleen was distinctly enlarged; the urine was normal; the leucocytes were 9000; the Widal reaction was positive. From this time the case gave the typical appearance of a moderately severe attack of typhoid fever in a child. July 12th there was a moderate eruption of rose-spots on the abdomen; the temperature ranged between 104° and 105°, and she responded poorly to antipyretic measures. July 17th she complained of so much soreness in the mouth that it was necessary to feed her through a tube. The pulse was rapid and weak, and the temperature still remained elevated. Her condition remained exceedingly serious until July 28th, when such distinct improvement occurred that she was able to take milk by the mouth. Investigation of the mouth showed at this time some necrosis of the upper jaw on the right side, and the canine tooth in the necrotic area was loosened. July 30th a culture was taken from this necrosed area, which in the meantime had extended slightly. The leucocytes were 9000. August 3d it was noted that the necrosed area had not increased. The temperature was lower and the patient was obviously better. August 7th the report returned from the Pepper Laboratory stated that the diphtheria bacillus was present in the necrotic area, and an injection of anti-diphtheritic serum was immediately given. August 8th a

discharge was noted from the right ear. On this day a second culture from the mouth was positive, and a culture from the discharge from the ear also developed diphtheria bacilli. August 9th, 500 units of antitoxin were administered. A loose tooth fell out. August 11th another tooth fell out and a culture was made from the cavity, diphtheria bacilli being found. On this day the leucocytes were 10,900. August 15th the necrosed area was healing rapidly; the temperature was normal, and the patient had apparently recovered. August 20th a culture from the mouth was negative, and the patient was discharged cured two days later.

At the time that this patient was in the hospital another brother was also in the children's ward suffering from typhoid fever. He did not develop necrosis of the jaw, and unfortunately cultures from the throat were not made.

Briefly summarized, the interesting features of these cases are that two brothers and a sister from the same family were admitted to the hospital in a short period, all suffering from typhoid fever. The previous summer one of the brothers had had an attack of diphtheria from which he had apparently recovered completely. Toward the end of the typhoid infection the sister and the brother who had had diphtheria, developed necrosis of the jaw, followed in the brother's case by typical cancrum oris. Diphtheria bacilli were obtained from the necrotic and gangrenous areas in both cases.

Noma is an exceedingly rare complication of typhoid fever. In the majority of text-books that I have consulted it is not even mentioned. Dr. Keen states that in 1700 cases with surgical complications that he analyzed, noma was recorded 9 times. Five of the patients died; 3 recovered; and the termination in one was not given. As these were selected cases, it is evident how very unusual noma really is. Walsh, however, in his analysis of the statistics collected by Hildebrandt and Perthes, notes that noma followed typhoid fever in 26 of 133 cases, a proportion that is not, he says, in accordance with his own experience. Worochinin[1] reports a great number of cases of noma, 46 of which were treated in the hospital; and in 31 of these the associated or predisposing disease could be determined. In these 31, 2 followed typhoid fever, and it is interesting to note that in both the patients had complained of sore-throat. In one of these the condition was described as faucial diphtheria, and in the other as a cachectic angina. Of course, the majority of these cases were observed before the discovery of the Klebs-Loeffler bacillus. Of the remaining 29 cases, one followed scarlet fever and diphtheria; 2 were associated with ulcerative stomatitis; and one, a case of noma of the vulva, occurred in a girl of three years, who was said to have suffered from diphtheritic vulvitis. Only 3 of 22 cases in which the results were given recovered.

[1] Jahrbuch für Kinderheilkunde, 1887, vol. xxvi. p. 167.

The first article in which attention was called to the possibility that noma, in some cases at least, is due to the diphtheria bacillus, was that of Freymuth and Petruschky.[1] Curiously enough, this paper attracted very little attention at the time. They observed 2 cases: one in a girl, aged three years, who had an attack of measles followed by noma of the vulva and of the adjacent parts. The Klebs-Loeffler bacillus was found in the gangrenous areas, and the patient subsequently developed diphtheria of the fauces. Antidiphtheritic serum was given in large and repeated doses, and the patient recovered. Petruschky had also discovered the diphtheria bacillus in a case of gangrene of the foot, and has even found one similar to it, if not identical with it, in measles. In discussing the bacteriology of the case he notes that the culture obtained was only slightly pathogenic for guinea-pigs, that is to say, it caused death in about ten days, with the characteristic changes, such as swelling of the adrenals. In the same year[2] they reported the second case occurring in a boy, aged eight years, who, in the sixth week of typhoid fever, developed stomatitis, followed by typical noma. The Klebs-Loeffler bacillus and the pseudodiphtheria bacillus were found, and the former was only slightly pathogenic for guinea-pigs. The authors express the opinion that probably the Klebs-Loeffler bacillus would be found in many cases of noma if it were sought for, particularly as diphtheria and measles and noma and measles are very frequently associated.

The next publication on the subject was that of Passini and Lenier,[3] who reported a case occurring in a boy, aged eight years, living under unfavorable hygienic conditions, who developed a blister upon the cheek, then a black spot, and finally typical noma. When brought to the hospital he was moribund, but nevertheless the Klebs-Loeffler bacillus was found in the gangrenous area. At the autopsy tuberculosis of nearly all the organs was found.

Perhaps the most important contribution to this subject is that of Walsh.[4] Being familiar with the paper of Freymuth and Petruschky, and having an opportunity to study 8 cases of noma, he examined them for the diphtheria bacillus, and found it in all cases. In addition, he made cultures from 15 cases of simple ulcerative stomatitis, all of which were negative. Five of the cases of noma had recovered from measles; 1 from diphtheria, and 2 apparently occurred spontaneously. In addition to these articles it may be mentioned that Nicolaysen[5] found in 2 cases of noma a non-pathogenic bacillus resembling the diph-

[1] Deutsche medicinische Wochenschrift, 1898, p. 232.
[2] Ibid., 1900.
[3] Wien. klin. Woch., 1899, p. 734.
[4] Proceedings of the Pathological Society of Philadelphia, June, 1901, p. 179.
[5] Norsk. Mag. for Lägevid, 1896, quoted by Klautsch, Archiv für Kinderheilkunde, 1899, vol. xxvi. p. 245.

theria bacillus. In view of the observations of Petruschky regarding the diminished virulence of the diphtheria bacillus associated with noma, it is possible that Nicolaysen was actually dealing with the true germ. Aside from the diphtheria bacillus, the number of micro-organisms that have been discovered is considerable. Among the most important are the long, thread-like bacilli found by Grawitz,[1] and observed in smear preparations from the mouth of Case I., and a very straight, non-motile bacillus with round ends, found in 3 cases by Babes and Zambilovici,[2] and by Guizetti.[3] The latter micro-organism produced gangrene in rabbits.

A hypothetical history of the cases I report might be given as follows: The first patient contracted diphtheria, from which he recovered, but the diphtheria bacilli remained in the buccal cavity. At the same time, his sister either had the same disease in a mild form, or else received diphtheria bacilli from her brother in a state of such diminished virulence that they were not capable of causing inflammation. In both cases, however, the micro-organisms retained sufficient pathogenicity to produce necrosis and gangrene in tissues whose vitality was lowered by another grave infectious process. That the germs were not very pathogenic was proved by the fact that two guinea-pigs into which pure cultures were injected became very sick for a few days, but did not die.[4]

These cases, I believe, are important for the following reasons: In the first place, they tend to confirm the opinion that has been frequently urged, but has not acquired sufficient adherents, that in all cases of sore-throat an examination for diphtheria bacilli should be made, and that if they are found a prophylactic injection of antidiphtheritic toxin should be given to the patient. In the second place, they seem to show that one of the causes of noma is the diphtheria bacillus; that it acts as such a cause usually in the presence of some other infectious disease, particularly in patients whose physical condition is, aside from the infectious disease, bad; and that as a general rule the diphtheria bacillus in these conditions is of diminished virulence, and not capable of producing the characteristic signs and appearance of true diphtheria.

In conclusion, I desire to express my thanks to Professor J. P. Crozier Griffith for his generous permission to report the second case, and to Dr. Levi, my resident physician, for his assistance in the study and observation of the first case.

[Since the above article was written the admirable report of an epidemic of noma by Drs. Blumer and McFarlane has appeared in THE

[1] Deutsche medicinische Wochenschrift, 1890.
[2] Annales de l'Institut de Pathologie du Bucarest, 1895. [3] Policlinico, 1896.
[4] Unfortunately, Dr. Kneass was absent from the city, and further experiments could not be made.

AMERICAN JOURNAL OF THE MEDICAL SCIENCES for November, 1901. The authors believe that noma is most frequently due to a thread-like organism of the leptothrix type that does not grow upon ordinary media. The diphtheria bacillus was apparently excluded as a causative factor in their cases.]

THYROIDITIS COMPLICATING TYPHOID FEVER.*

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INFLAMMATION of the thyroid gland is not uncommon in those localities where goitre is endemic. In such cases it occurs usually as an acute condition, in a gland the seat of previous hypertrophy. This is known as strumitis acuta. Inflammation of a previously healthy gland, however, is rare. Bardeleben, in Eulenberg's *Encyclopædia*, said he had never seen a case, and both Paget and Semon similarly expressed themselves when their attention was drawn to Barlow's case, though Semon has since reported one coming under his own observation.

Lücke,[1] in Pitha and Billroth's *Surgery*, in his article on "Acute Inflammation of the Thyroid," gives a concise description of the inflammations of the previously healthy gland, and divides them into: 1, idiopathic; 2, traumatic; 3, metastatic. The idiopathic form, he says, is extremely rare, occurs especially in young people about the age of puberty, and is etiologically very obscure. According to Godlee,[2] the following varieties are met with: 1. Idiopathic, with spontaneous subsidence. 2. Epidemic, as described by French military surgeons. 3. Septic form. This classification takes no cognizance of the traumatic form.

Mygind[3] divides thyroiditis into the simple and suppurative, the former terminating in resolution, and the latter in abscess. Though this grouping fails to take any note of the etiological factors, it seems to me the best, if for no other reason than that it obviates the use of the term idiopathic. Admittedly rare, as already stated, Mygind was only able to collect from the literature seventeen cases of acute inflammation occurring in a previously healthy gland, and ending in resolution.

The condition occurs most commonly in females, according to Mygind,[4] particularly between the second and third decades; rarely in previously healthy persons and without any apparent cause, sometimes as a result of traumatism, and finally, in the so-called metastatic cases, during or

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immediately following an attack of typhoid fever, rheumatic fever, diphtheria, influenza, malaria, erysipelas, puerperal fever, sepsis, orchitis, or parotitis. The traumatic and metastatic forms almost invariably terminate in suppuration, except those possibly occurring in the course of rheumatic fever or malaria. Sajous[5] says that the uterine, menstrual, and climacteric disorders tend to cause congestion, which, often repeated, may give rise to active inflammation of the thyroid gland. French writers[6] on military surgery have reported many epidemics of acute enlargement of the thyroid occurring among soldiers in garrisons, and Viry and Richard, who reported the Belfort epidemic of 1877, claimed that the epidemic disease is an acute specific one, having no relation whatever to ordinary goitre. The French writers further speak of the resemblance of this form of thyroiditis to mumps.

Semon has reported a case of thyroiditis alternating with mumps. Most commonly, rheumatic or typhoid fever is the immediate antecedent of thyroiditis. Moliere,[7] of Lyons, in 1873, was the first to record a case of acute thyroiditis supervening on acute rheumatism. It developed when all the joints had ceased to be painful.

Vulpian,[8] in 1877, reported a case following arthritis of the fingers, associated with generalized pain. Raymond[9] another following multiple arthritis. Barlow's[10] case was a child of three years, the youngest I have seen recorded. The boy had had a mild attack of erythema nodosum and was almost well when the thyroid became acutely inflamed; fever, dysphagia, and stiffness of the neck supervened, reached its acme in four days, and subsided without suppurating after a course of about ten days. Angel Money,[11] in the discussion of Barlow's paper, spoke of a case of a young woman who had had repeated attacks of rheumatism with heart disease and erythema papulatum and marginatum, who, when convalescent from one of the attacks, acutely developed inflammation of the thyroid. It soon resolved. Given[12] reports the case of a nurse, aged twenty-three years, who had just recovered from an attack of rheumatism when she suddenly fell ill with general malaise, pain and swelling of the neck in the region of the thyroid, temperature 105.3°, dysphagia, dyspnœa, inspiratory stridor, tenderness over thyroid, and subsequent resolution without suppuration.

It is, however, as a complication, or more frequently as a sequel of typhoid fever that we usually meet with acute thyroiditis, and in the majority of cases it goes on to suppuration, though it must not be lost sight of that even in typhoid the condition is rare, as the following statistics show:

Among 349 autopsies in Hamburg, Curschmann[13] did not see a single case, and only twice clinically during the large epidemic in that city. In Leipzig, too, he says it is quite rare. Walther,[14] who published the only case of thyroiditis typhosa occurring in Curschmann's clinic, had

previously shown that of 73 collected cases of acute strumitis and thyroiditis, 40 were associated with typhoid fever. The relative frequency of thyroiditis in Switzerland explains the findings of Griesinger[15] and Liebermeister.[16] The former, four times in 118 autopsies, the latter, 15 times among 1700 patients, in 6 of whom suppuration developed. Among the complications of typhoid fever, Topfer[17] mentions 3 cases of abscess of the thyroid in 927 autopsies.

The immediate exciting cause of thyroiditis occurring during the course, or as a sequel of typhoid fever, is worthy of some attention. Is the typhoid bacillus ever pyogenic, or are the various grades of inflammations due to the ordinary pus-producing organisms, especially the staphylococcus and streptococcus? There is no longer any doubt that the bacillus of Eberth assumes pyogenic properties in certain cases. This was definitely proved, first by Fränkel,[18] who recovered it in pure culture from a case of encysted peritonitis; but two years before, Freund[19] claimed that inflammation of bone occurring during convalescence from typhoid was due to the specific action of the typhoid bacillus. Ebermaier,[20] among 8 cases of typhoid periostitis, observed 2 which went on to suppuration, and recovered from the blood and tissues of the affected part a pure culture of typhoid bacilli; Valentini[21] the same from a case of empyema complicating typhoid fever; Achalme[22] in osteoperiostitis of the tibia occurring in the beginning of convalescence from the same disease. Chantemesse and Roux[23] have experimentally produced pus by injecting rabbits with a virulent typhoid culture, as has Honl,[24] who tabulated his results as follows:

1. When injected into certain parts of the animals, typhoid bacilli may cause pus.
2. Toxins and old cultures have pyogenic properties.
3. The concentration of the culture has no influence on increasing the pyogenic function.
4. Bacilli from abscesses disappear very rapidly.

In the course of typhoid, especially toward convalescence, various inflammatory affections may arise, due to invasion of germs with pyogenic properties, only rarely to the typhoid organism alone. Honl[25] has summed them up in the following order of frequency:

a. Secondary invasion of pyogenic cocci.

b. Secondary microbiotic mixed infection, *i. e.*, through cocci or other microbes plus typhoid bacilli.

c. Exclusively through the bacillus of Eberth.

Honl states that in the Pathologico-Anatomical Institute of Prague, in ten years, 1883 to 1893, 165 autopsies were performed on typhoid cases, in 16.39 per cent. of which some purulent process had been the cause of death. Of more direct interest is the finding of Spirig,[26] who was the first to cultivate typhoid bacilli in a case of strumitis; he also

found staphylococci. Chantemesse,[27] from the pus of a case of suppurative thyroiditis complicating the defervescent stage of typhoid fever, recovered the typhoid bacillus with the ordinary organisms of suppuration. The bacillus of Eberth was also found in typhoid thyroiditis by Lichtheim-Tavel[28] and Jeanselme.[29] The mixed infections are, as a rule, the most serious; indeed, they are often but part of a general sepsis.

The case which I wish to present did not go on to suppuration, but illustrates the simple type of Mygind. The patient, a man, aged forty-two years, so far advanced in convalescence from typhoid as to have reached a normal temperature, began to complain of difficulty in swallowing. The thyroid gland became enlarged quite acutely, the right lobe being more involved than the left, it was quite tender on palpation, giving a sense of semi-fluctuation. Soon after the onset he had a slight chill, his temperature became moderately elevated, he complained of a feeling of constriction about his throat, dysphagia became more pronounced, indeed, it was the most troublesome feature of the attack; slight dyspnœa supervened, and owing to the pain in the neck, his head was held rather rigidly. For three days he was scarcely able to swallow liquids, but with the subsidence of the tumefaction all manifestations gradually vanished, the process terminating in resolution after about ten days. Such attacks are usually sudden in onset.

When the left lobe of the thyroid is especially implicated, dysphagia is apt to be more pronounced, owing to greater opportunity for pressure because of the left lateral deviation of the œsophagus. Dyspnœa is seldom severe and rarely associated with stridor. Hoarseness is exceptional. It is due to pressure on the inferior laryngeal nerve, which is situated between the lobes of the gland and the œsophagus. Mygind's[30] is the only case of the kind reported, though Galtier speaks of the possibility of such a condition. In Mygind's case complicating facial erysipelas in a man of thirty-eight years, the great hoarseness lead to an examination of the larynx. The mucous membrane was found inflamed and swollen, the left vocal cord being fixed in the cadaveric position during deep respiration, and only moved a little toward the median line on phonation. It is very rarely that pressure on neighboring vessels gives rise to any symptoms. Of course, all symptoms are apt to be more pronounced in suppurative cases, but very seldom do threatening symptoms supervene.

Griesinger[31] has published a death from suffocation, and Forgue[32] another, in the latter case due to perforation of a thyroid abscess into the trachea. Luigi Porta,[33] in an extensive article on diseases and operations of the thyroid gland, speaks of the possibility of death by erosion of a large vessel, and he further gives a most grave prognosis in those cases of deep-seated abscesses in which the pus finds its way into the mediastinum or pleura. The abscess may be single or multiple,

but in any case is apt to involve one lobe more than the other. Of the ten case of thyroiditis complicating typhoid fever collected by Keen,[34] in all but one suppuration occurred. In four of them the gland was previously hypertrophied. In seven the attack began during convalescence, *i. e.*, after the third week, in two as late as the seventh or eighth week. This is characteristic of glandular involvement in typhoid, be it of the testicle, ovaries, parotid, or thyroid, that it is a consecutive or secondary manifestation, and belongs to the period of decline and convalescence. Tavel and Laveran[35] have shown that it may very rarely develop with the onset. One very curious case published by Kummer and Tavel and quoted by Keen[36] was as follows: Suppuration occurred in a goitrous gland, from the pus of which Eberth's bacillus was recovered. No typhoid bacilli were found in the stools, nor any other evidence of typhoid infection.

The prognosis is good, threatening symptoms are unusual, and fatalities are among the greatest rarities, as has been stated. The consensus of opinion is that goitre never results, but Luigi Porta[37] says hypertrophy may occur in consequence of unresolved inflammation. I do not know of any recorded case.

The course of the attack is generally short, ten to fourteen days.

The treatment of the simple variety of thyroiditis consists merely in the use of ice locally. If suppuration is impending it may be hastened by hot fomentations, and an incision should be made as soon as evidence of pointing or fluctuation occurs. If the gland is the seat of pre-existing hypertrophy, it would be advisable to extirpate the suppurating half, as Kummer did. This would be done, of course, at some subsequent period when the patient had well advanced in convalescence.

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CAUSE OF DEATH IN ANEURISMS OF THE THORACIC AORTA WHICH DO NOT RUPTURE; REPORT OF FIVE CASES.

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Owing to the frequency with which incomplete post-mortem examinations have been made in cases of thoracic aneurism, statistics are not entirely satisfactory. Hare and Holder[1] have collected from medical literature 953 cases of aneurism of the arch of the aorta. Among their conclusions these writers make the observation[2] "that in a large proportion of cases death did not ensue from rupture but from pressure by the growth." In another place they say:[3] "Death occurs as a result of aneurism of the aorta either from rupture of the aneurismal sac, from pressure of the sac upon important nerves and bloodvessels, or from the secondary changes which take place in these tissues and in other vital organs as a direct or indirect result of such pressure."

From various data in their article I have made the following tabulation of the number of cases in which it was stated that death occurred without rupture.

Ascending arch,	570	. . .	Unruptured,	93 = 16	per cent.
Transverse "	104	. . .	"	20 = 19	"
Descending "	110	. . .	"	17 = 15	"
Unclassified,	169	. . .	"	24 = 14	"
Total,	953	. . .	"	154 = 16	"

I have also made a tabulation of the cases in which rupture is definitely stated to have occurred.

Ascending arch,	570	. . .	Ruptured,	339 = 60	per cent.
Transverse "	104	. . .	"	39 = 38	"
Descending "	110	. . .	"	72 = 66	"
Unclassified,	169	. . .	"	74 = 44	"
Total,	953	. . .	"	524 = 55	"

Browne,[4] in a series of 173 cases, found that only 84, or 49 per cent., were due to rupture, a smaller percentage than that among the cases reported by Hare and Holder. Sibson[5] made an analysis of 584 reported cases of aortic aneurism and of 296 museum specimens. Of the total number of 880 cases 703 were instances of aneurism of the thoracic aorta. Of these latter 358 ruptured, or 51 per cent.

Hare and Holder	953 cases.	Ruptured,	524 = 55	per cent.
Sibson	703 "	"	358 = 51	"
Browne	173 "	"	84 = 49	"
Total	1829 "	"	966 = 53	"

The inference seems almost as reliable that nearly one-half of the cases of aneurism of the thoracic aorta die without rupture of the sac.

Turning, now, to the analysis of the 154 cases collected by Hare and Holder, of which it was stated that death was not due to rupture, I have made the following tabulation of the causes assigned for death by the various observers: *

Obstruction to air-passages		66
Dyspnœa	26	
Suffocation	10	
Pressure on trachea	27	
Pressure on bronchus	2	
Œdema of larynx	1	
Exhaustion		50
Affections of lungs and pleura		28
Apoplexy of lung	3	
Congestion of lungs	2	
Pneumonia from pressure	2	
Pneumonia	1	
Pleuropneumonia	1	
Abscess of lung	1	
Bronchitis	2	
Serous pleural effusion	14	
Pleurisy	2	
Pericardial affections		8
Serous pericardial effusion	7	
Pericarditis	1	
Pressure on the vena cava superior		1
Collapse		1
		154

It is clear at a glance that this information is vague and unsatisfactory. The largest single item is death from exhaustion, comprising practically one-third of the cases. In too many of these cases, I fear, the reporter thoughtlessly said " exhaustion " without any special effort to see if there were a better explanation. Suffocation, credited with ten deaths, leaves the mind guessing as to the exact way in which it was caused. Pressure on the trachea, credited with twenty-seven deaths, is an adequate cause of death if the pressure is extreme, but moderate

* It may be noted again that the faults in this table are due to the imperfect report of cases, and not to the excellent compilation made by Hare and Holder.

pressure does not necessarily cause death, though doubtless it may be one of the factors in producing that result. Dyspnœa is fatal not in itself, but from the condition of which it is a symptom. It may signify trouble in the respiratory organs, in the circulation, or irritation of certain parts of the nervous system. The twenty-six deaths caused by dyspnœa might certainly be more carefully analyzed. It is a noteworthy fact that any complication involving the heart is not once mentioned specifically as a cause of death in these cases, though recognized by all the leading authorities as holding an important place in this connection. Only one case is mentioned of pressure on the great vessels about the heart—another well-recognized factor in the fatal results. Undoubtedly more careful observation would have shown that many of the cases classed under the heads of exhaustion, dyspnœa, suffocation, etc., were really deaths due to disturbance of the circulatory apparatus. It is not an exaggeration to say that many of these supposed explanations do not explain, and I think it is evident that there is need of a closer scrutiny of the fatal cases which do not rupture and a more thoughtful consideration of the real cause of death.

Let us consider briefly some of the problems to be thought out in this connection. We may divide the cases into three classes:

I. Cases in which an aneurism of the arch of the aorta exists, but in which it is not the important factor in the fatal result.

II. Cases in which death ensued from disease or disturbance of the circulatory system not caused by direct pressure of the aneurismal tumor.

III. Cases in which death was due, directly or indirectly, to pressure of the aneurismal tumor upon organs of vital importance.

Class I. comprises those cases which die from some intercurrent disease whose origin cannot properly be ascribed to the aneurism. Diseases of other parts than the thoracic organs or circulatory apparatus need no special discussion, and even some diseases of the thoracic organs may be so obviously due to other causes than the aneurism that its existence is plainly an unimportant incident. On the other hand, it will not be easy in other cases to decide how far the aneurism may have been a contributing factor in the causation of such disease. This is illustrated by the following case:

Case I.—Male, aged fifty-one years, stationary engineer. His family history and previous history are unimportant, except that when twenty years old he was thrown from a locomotive and injured his back so that he lost the use of his legs for one month, and that he had two attacks of pneumonia nine and seven years ago. No history of syphilis could be obtained. Alcohol was not used to excess. On December 31, 1897, he was assaulted by two men, strangled, and beaten until insensible. It is probable that the strangulation, together with the violence of his struggles, may have been the immediate cause of the giving way of the

arterial wall. Certain it is that as a result he suffered from broken cardiac compensation for several months.

The more serious symptoms of his present trouble date from April 1, 1901. His first serious attack of dyspnœa occurred on April 19th. This attack resembled angina pectoris in the nature of its distress and anxious suffering. It also suggested to the attending physician emphysema with an asthmatic attack. There was marked respiratory dyspnœa, "rattling breathing," and stridor. There was very little cough. On the night of April 25th, after drinking a certain amount of liquor, while walking on the street, he "felt queer," rapidly choked up, lost consciousness, and did not awaken until some hours later at the hospital.

At this time the physical examination showed the pupils equal, with normal reaction. Pulse was irregular, slow, of good volume and tension. The walls of the radial arteries were sclerotic. The right border of cardiac dulness was 3½ cm. to right, and the left border 10½ cm. to left of the median line; upper border at the third rib. The apex was in the fifth interspace, 10 cm. to left of median line. The heart sounds were faint. No murmurs were detected. The lungs were negative. No signs of aneurism were detected. He improved and left the hospital April 27th at his own request.

On April 28th he had two attacks of dyspnœa, similar to his first attack. On April 29th he had two attacks; on April 30th one attack. His next attack was on May 12th. Four attacks followed in the next two days, and the final attack began on the night of May 15th.

He was brought to the hospital in a semi-conscious condition. The peculiar crowing respiration and brassy cough at once attracted attention, and suggested the possibility of aneurism. A tracheal tug was found. Dulness in the second interspace extended 5 cm. to the right of the median line, but there was no impulse, thrill, or bruit detected. The right radial pulse was entirely obliterated, the left was regular and of only fair strength and volume. The heart was the same as at the previous examination, but weaker. Percussion showed fair pulmonary resonance throughout the chest. Auscultation showed exaggerated, crowing respiration, with many coarse bubbling and fine moist râles. He did not improve, the pulmonary œdema increased, and he died early the next morning.

The autopsy showed a sacculated aneurism of the transverse arch of the aorta, and a dilatation of the ascending arch; chronic endocarditis of the mitral valve; hypertrophy and dilatation of the left ventricle; general chronic passive congestion; arterio-sclerosis; lobar pneumonia; acute congestion, and œdema of the lungs.

The heart muscle appeared of good quality and showed no fatty changes microscopically. There was nothing noteworthy beyond a moderate thickening of the mitral valve and chordæ tendinæ and moderate hypertrophy and dilatation of the left ventricle.

The aortic orifice measured 8 cm. in circumference; one centimetre higher the aorta measured 9 cm., and six centimetres beyond the valve it measured 11 cm. At nine centimetres from the valve a sacculated dilatation began, extending backward from the transverse arch. The dilated aorta merged into the aneurismal sac without a definitely marked border on the side toward the heart. The distal edge of the sac had a definite rounded border. At this latter point the aorta measured 6 cm. in circumference, and continued of this size to the diaphragm.

The opening from the aorta into the sac measured 4 to 5 cm. in diameter, and 14.5 cm. in circumference. The circumference about the sac and aorta was 15 cm. The intima of the aorta and the lining of the sac showed irregular thickened patches, but no calcification.

The posterior wall of the sac was in contact with the trachea just above its bifurcation. The wall of the sac was eroded at this point over an area 4 by 2 cm., and here the rough tracheal wall and the cartilaginous rings could easily be felt. The tracheal wall was somewhat thinned at this place. The trachea was somewhat flattened anteroposteriorly, but at the post-mortem examination there was no definite bulging of the aneurism into the lumen of the trachea.

The cavity of the aneurism contained fluid blood except for a small amount of fresh red clot. The left pneumogastric nerve ran down the side of the sac anteriorly, but seemed perfectly free. The right pneumogastric nerve was not in direct relation to the sac.

The vessels of the neck arose from the anterior wall of the sac. The left carotid and subclavian arteries appeared normal. There was no opening to the innominate artery from the sac. Where the opening should be was a smooth, rounded thickening with a very small depression. The smallest probe could not find a passage. On opening the innominate artery from its distal end it appeared normal until within one centimetre of the sac, when it suddenly narrowed and was completely occluded by a firm, reddish-yellow thickening beneath the intima on one side. The arteries and veins of the right arm appeared normal and were filled with blood.

An area of acute lobar pneumonia occupied about the upper fourth of the lower lobe of the right lung. It also extended to a small area at the root of the upper lobe, but did not involve the middle lobe. The remaining portions of both lungs showed intense acute congestion and œdema. Death was evidently due to this congestion and œdema of the lungs, which in turn was caused probably by the toxins of the pneumonic inflammation. The stage of development of the pneumonia indicated that it was about the fourth or fifth day, which suggests that the series of attacks beginning May 12th were induced by the pneumonic process.

What part did the aneurism play in causing the death of this patient? It certainly was not the immediate cause, yet it is hard to believe that it did not have an important contributing influence. In the first place the recurrent attacks of extreme and prolonged dyspnœa must have materially affected the general condition and strength of the patient. This reduced the resisting power of the system of an individual who was easily susceptible to pneumonic infection, as shown by the history of two previous attacks of pneumonia. It would seem probable, also, that the obstruction to the passage of air to the lungs might have induced changes in the lung tissue, rendering those tissues more susceptible to invasion by the pneumococcus. Finally, after the pneumonic process started, the frequent attacks of dyspnœa from the pressure of the aneurism on the trachea must have had a bad effect upon the patient's vitality in fighting the pneumonia. It is not clear how far the

last attack of dyspnœa was due to the aneurism, and how far to the general acute congestion and œdema of the lungs. When he was examined at the hospital pulmonary œdema was already well established, but he also had the crowing respiration and brassy cough of aneurism.

In Class II., which includes cases of death from circulatory disturbance not caused by direct pressure on the bloodvessels, the chief doubt will arise in determining how far in cases associated with arterio-sclerosis death may have been caused by that disease rather than by the aneurism itself. Arterio-sclerosis may cause death independent of the aneurism, as a result of changes in the nutrition of vital organs, by rupture of the diseased arteries, by the plugging of the arteries from thrombosis or embolism, or by its combined effect on the work and the nutrition of the heart. If death occurs from embolism, its source may be the aneurism itself, since coagulation of blood is favored in the aneurismal sac, and the danger of embolism is thus increased. Or, again, the aneurism may be an important factor in causing death by its effect upon the heart.

What effect may an aneurism of the aorta have upon the heart? It has been claimed by some that it causes hypertrophy of the heart. Calvert[6] has shown that this is not necessarily the case. His conclusion is based on statistics gathered by Dr. Oswald A. Browne from the cases occurring in St. Bartholomew's Hospital in thirty years. Of 124 cases, 68 showed no hypertrophy; in 47 the hypertrophy could be satisfactorily explained by other causes; and in the remaining 9 hypertrophy could be explained as probably due to other causes. In arterio-sclerosis we have a very common cause of heart hypertrophy, so that in many cases the aneurism and the heart hypertrophy are to be regarded as common results of the same cause and not as one dependent upon the other. One form of aneurism, however, certainly has a direct effect upon the heart. I refer to aneurisms situated close to the aortic valve and involving a general dilatation of the aorta. If such an aneurism enlarges enough it will by its stretching cause dilatation of the aortic orifice, from which follows aortic regurgitation with its results. Case II. belongs to this group.

Case II.—Male, aged forty-four years, choreman, came to the out-patient department of the Boston City Hospital, February 18, 1899. Family history is unimportant. He had syphilis twenty-five years before. He had used alcoholic drinks moderately.

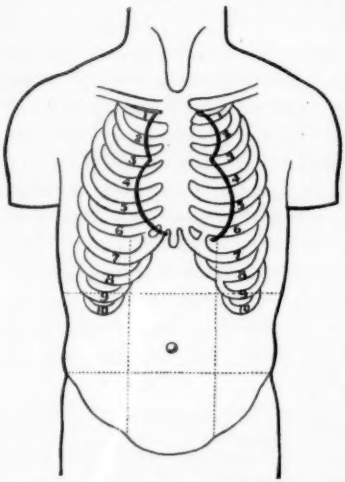
His chief complaint was of pain and a feeling of pressure in the præcordial region, increased by exertion. He had suffered from this about one week. His trouble, however, began at least a year before, at which time he began to have a severe boring pain in the back of the chest, together with dyspnœa on exertion. At the time of the examination he was not hoarse, but he had had several attacks of hoarseness in the last few years.

Inspection showed the pulsation of the carotid arteries more marked and jerky than normal. On the right side of the neck, just above the

inner end of the clavicle and also in the episternal notch, a marked pulsation was seen. Palpation showed that the pulsation in the episternal notch had a distinct lateral thrust from the right, indicating its origin from the innominate artery and not from the arch of the aorta. A thrill was plainly felt just outside the sternomastoid muscle above the clavicle. The right radial pulse was weaker than the left, but there was no perceptible difference in time. A slight but distinct tracheal tug was made out. The pupils were equal. The apex-beat of the heart was felt in the sixth interspace in the left mammary line.

Percussion over the lungs was normal. Relative dulness of the heart extended to the left mammary line, four and seven-eighth inches to the left of the median line, and on the right its border was one inch to the right of the median line. The area of dulness over the great vessels was increased in breadth. On the left, at the base of the heart, dulness extended two inches from the median line, diminishing to one and five-eighth inches higher up. On the right the distance was one inch from the median line at the base of the heart, increasing to one and three-eighth inches just below the clavicle. The line of dulness bounding the great vessels on the right, together with the abnormal pulsation beneath the inner end of the right clavicle, made the diagnosis of aneurismal dilatation of the innominate artery practically certain. The breadth of the area of dulness over the vessels just above the base of the heart, with the tracheal tug and the associated aneurism of the innominate artery, made it highly probable that an aneurism existed in the ascending and transverse parts of the arch of the aorta. Physical examination gave no signs of the involvement of the descending arch.

FIG. 1.



Fluoroscopic examination.
Fusiform aneurism of ascending, transverse, and descending arch.

Confirmation of the diagnosis was sought in an X-ray examination, which was very kindly made for me by Dr. F. H. Williams. The accompanying diagram shows the outline of the shadow cast by the heart and great vessels as shown by the fluoroscope.

The point of special interest in the X-ray examination lies in the shadow with a curved outline above the left base of the heart. This shadow extends upward three inches from the base of the heart, and outward to the left one and one-quarter inches beyond the origin of the great vessels. Distinct expansile pulsation was seen in this shadow just following the systolic contraction of the ventricle. It evidently shows an aneurism of the aorta extending from the transverse arch to the descending arch. A distinct outline of the innominate aneurism could not be obtained, perhaps owing to its relation to the shadow of the spine.

By the fluoroscopic examination we made certain the existence of an aneurism of the arch of the aorta, whereas before we had great probability rather than certainty; we also learned that this aneurism extended to the descending arch, of which fact the ordinary physical examination did not give even a clue, and we were able to outline the size of the heart more accurately than by percussion.

Auscultation of the lungs was normal. At the aortic area a systolic and a diastolic murmur were heard. The systolic murmur had its maximum intensity at the aortic area. It was transmitted upward along the aorta and could be heard faintly in the left carotid and subclavian arteries. Just at the inner end of the right clavicle the systolic murmur was markedly reinforced, and had an intensity even greater than at the aortic area (a condition which was interestingly explained by the post-mortem findings), and the murmur was quite loud in the right carotid and subclavian arteries. The diastolic murmur could not be heard in the left carotid or subclavian arteries, but was faintly audible in the corresponding vessels of the right side. Its maximum intensity was at and just above the aortic area. It was not carried downward beyond the anatomical position of the aortic valve (the junction of the third left costal cartilage with the sternum), and at this point the second sound became distinct and somewhat accentuated. A slight systolic murmur was also heard at the apex, but was not transmitted far in any direction. It was not clear whether this was the aortic systolic murmur transmitted or not.

The diagnosis was aneurism of the aorta, involving the ascending, transverse, and descending portions of the arch; aneurism of the innominate artery, and regurgitation at the aortic valve. The patient was ordered to take iodide of potash, beginning with ten grains three times daily, and he was especially cautioned against sudden or marked exertion.

On February 23d, five days later, he returned. He felt much better. The pain in the shoulders and upper chest was much less, but he still complained of the præcordial pain. Physical examination was the same, except that the radial pulse was fuller and apparently stronger. His condition was apparently improved. The dose of iodide of potash was increased to fifteen grains.

This observation was made about 1 P.M. The patient was found not long afterward on a door-step not far from the hospital. He looked sick and distressed, and was very weak. He told the person who found him that he had been at the hospital for treatment. This person assisted him to arise, but finding he was too weak to walk, he left him and hurried to the hospital. An ambulance was sent at once. The patient was carried directly to the ward, and was seen immediately by the house physician. He was in collapse, pale, and with no radial pulse. His mind was clear. He did not respond to stimulants, but grew steadily worse and died in about ten minutes. This was about one hour after he was seen at the out-patient department. It was naturally supposed that the aneurism had ruptured.

At the autopsy the following pathological conditions were found: Fusiform aneurism of the aorta and of the innominate artery; general arterio-sclerosis, with calcification and atheroma; general chronic passive congestion; œdema of the lungs.

The aneurism had not ruptured. The heart was enlarged, somewhat

distended with blood, and the right auricle was especially full and prominent, extending further to the right than normally. The aneurismal sac was not tensely distended, but rather relaxed. The aneurism was of the fusiform type, involving the whole of the arch. There was also a fusiform aneurism of the innominate artery, extending from its origin at the aorta to its bifurcation.

The circumference of the aortic valve was 8.5 cm.; six centimetres above this the circumference of the aorta was 13¼ cm.; at the innominate artery it was 13 cm; at the left subclavian artery, 10 cm., and at the first intercostal branches, 7 cm. The opening into the innominate artery had slightly thickened walls and measured 1.1 cm. in diameter. The artery gradually increased in size as it ascended, until it reached a diameter of 2½ cm. From this point the enlargement gradually diminished to the division into the carotid and subclavian arteries, which were both normal in size. There was a marked general arteriosclerosis, with calcification and some atheroma, involving the whole extent of the aorta.

The autopsy, then, thoroughly confirmed the diagnosis, and showed that the extent of the aneurism had been accurately recognized. Moreover, the shape of the innominate aneurism gave an adequate explanation of the peculiar intensity of the systolic murmur near the clavicle, namely, that a new murmur was here created as the blood rushed through the narrow opening into the larger space of the dilated innominate artery beyond.

Death came from the cardiac complication. The existence of aortic regurgitation had been recognized clinically by the aortic diastolic murmur, the enlargement of the left ventricle downward and outward, and the characteristic jerky pulsation of the carotids. At the autopsy the aortic cusps were practically normal, but there was dilatation and slight hypertrophy of the ventricle, and the aortic orifice was somewhat stretched, sharing in the dilatation of the aorta just above. This is shown by comparing the measurements of this case with the normal measurements (Mallory and Wright[7]).

	Normal	Case II.
Circumference of aortic orifice,	7.7– 8 cm.	8.5 cm.
" mitral "	10.4–10.9 "	10 "
" pulmonary orifice,	8.9– 9 2 "	8 "
" tricuspid "	12 –12.7 "	12 "
" ascending aorta,	7.4 "	8.5–13.25 "
" pulmonary artery,	8 "	?
Thickness of left ventricular wall,	0.7– 1 "	1.2 "
" right " "	0.2– 0.3 "	0 4 "

Here we find the measurements of all other orifices corresponding pretty closely to the minimum figures of the normal table, while we find the aortic orifice nearly one centimetre larger than the minimum figure of the normal table.

The autopsy also showed a general chronic passive congestion of the organs. This suggests that the systolic murmur heard at the apex was due

to mitral regurgitation. This regurgitation was secondary to the dilatation of the left ventricle, as the mitral curtains and chordæ tendinæ were normal. The œdema of the lungs, the distention of the cavities of the heart, and the clinical story of the rapid failure and death all point to failure of the heart muscle as the immediate cause of death. The muscle seemed to be in good condition, and microscopical examination failed to find fatty degeneration. The coronary arteries were patent. We have to fall back on the well-known clinical fact that death is apt to ensue suddenly in aortic regurgitation—a fact for which we are not yet always ready to furnish a satisfactory explanation. We are apt to think of death from aortic regurgitation as more rapid than this, which lasted somewhere between one-half and one hour. But Balfour[8] has called attention to the fact that ingravescent asystole occurs in aortic regurgitation, and may even be much more prolonged than in this case.

Our story in this case would seem to be clear, and the sequence of events as follows: Syphilis, arterio-sclerosis of the aorta, fusiform aneurism of the arch, resulting dilatation of the aortic orifice, aortic regurgitation, dilatation of the left ventricle, mitral regurgitation, general passive congestion, and death from asystole accompanied by œdema of the lungs. The responsibility of the aneurism for the death is indirect.

This case illustrates so well the view of fusiform aneurisms held by Sir Douglas Powell that I will quote a few of his remarks on the subject. He says:[9] "Clinically the phenomena characteristic of aneurism are scarcely ever observed except in association with the sacculated form, the so-called 'fusiform aneurism' being merged, as regards prognosis and treatment, in the class of heart disease with which they are most closely connected. These general dilatations involve, for the most part, the first part of the aorta, and are associated with valvular lesions and secondary cardiac dilatations and hypertrophies; they give rise, with rare exceptions, to no pressure signs; they do not tend to cause death by rupture or by compression of vital parts, but rather through cardiac failure, angina, or syncope. The treatment is that appropriate to heart disease."

Class III. includes by far the greatest number of cases. It is true in the vast majority of cases of aneurism of the aorta that if death is not due to rupture it is due, directly or indirectly, to pressure.

In its course from the left ventricle to the diaphragm the thoracic aorta lies in close relation to many important structures. It is worth while, in the study of the effects of pressure by the aneurismal sac, to briefly recapitulate those organs which may be affected.

The origin of the aorta is embedded in the structures at the base of the heart. In front lie the conus arteriosus of the right ventricle and the auricular appendage of the right auricle. The right auricle and

the vena cava superior are in close contact on the right. Around the left the pulmonary artery winds from the front, while behind lie the left auricle and the right pulmonary artery.

Just above the heart the edges of both lungs separate the ascending arch of the aorta from the sternum in front. The pulmonary artery is on the left. Behind and below are the right branch of the pulmonary artery and the root of the right lung. On the right lies the vena cava superior and the right lung. It should be remembered that the pericardium extends up on the aorta from one to one and one-half inches above the base of the heart.

The relations of the transverse arch are perhaps the most important of all. The ascending arch has a certain freedom of movement, and after it emerges from the structures at the base of the heart it may become considerably enlarged without serious results to surrounding organs—it displaces rather than compresses them. The transverse arch has much less freedom of movement, its relation to surrounding parts is closer, and these parts cannot readily be moved aside. Enlargement here causes compression rather than displacement. Symptoms of pressure, therefore, develop early in the process of enlargement.

The transverse arch passes backward and to the left in front of the trachea and above the left bronchus. The bifurcation of the pulmonary artery is beneath the anterior part of this section of the aorta. Above lies the left innominate vein. Further back than the trachea lie the œsophagus and thoracic duct. The relations of the nervous structures to this part of the aorta are important. The deep cardiac plexus lies behind the aorta, between it and the trachea, and above the bifurcation of the pulmonary artery. This is the main centre for the nerves distributed to the heart. The superficial cardiac plexus lies between the aorta and the right pulmonary artery. The left recurrent laryngeal nerve passes down in front of the aorta, passes backward beneath it, and ascends behind it and in front of the trachea. In front also pass the left vagus and phrenic nerves. From the transverse arch are given off the innominate, the left carotid, and the left subclavian arteries.

The descending arch passes backward and downward to the left of the trachea, behind the root of the left lung, to the left anterior aspect of the dorsal vertebræ. As it passes downward the œsophagus and thoracic duct lie on its right. The left pleura and lung lie to the left and anteriorly. The descending arch of the aorta and the thoracic aorta below are held firmly in place by the intercostal branches which are given off from the aorta.

We may now turn our attention to the ways in which pressure from an aneurism upon the organs surrounding the aorta may contribute to a fatal result. It is clear that the results will depend partly upon the

size of the aneurism and partly upon the vital importance of the organ or organs pressed upon. It is also manifest that the size of the aneurism is of less importance than the place where the pressure is exerted. A striking case to illustrate this point is reported by Irvine.[10] It was a case of double aneurism of the arch of the aorta. A relatively large aneurism of the ascending arch caused very little trouble, whereas death came because a small aneurism of the descending arch compressed the left bronchus and induced extensive changes in the left lung.

Before considering the effect on individual structures, however, let us note that death may come from the gradual wearing out of the patient's strength without any extreme pressure on vital organs. When an aneurism enlarges enough to press upon peripheral nerves pain results. Pain may also be of the nature of "referred pain," as in cardiac disease. Pressure on these nerves may also cause dyspnœa or cough, especially of a paroxysmal type. The suffering from the pain, the dyspnœa and cough, and sleeplessness due to any of these factors are the main causes of death in some cases, and such death may be properly called "death from exhaustion."

Pressure on the nerves may contribute to the death in other ways. Spasm of the glottis may result reflexly from irritation of the vagus nerve. And Gibson[11] says the same result "may possibly be an early symptom of interference with the motor nerve of the larynx," the recurrent laryngeal. To what extent pressure on the cardiac nerves or ganglia may exert an inhibitory or paralytic effect on the cardiac muscle is not perfectly clear. It is mentioned by some writers, and it may be a possible explanation of some of the cases where the most careful post-mortem examination fails to reveal any adequate explanation. This subject needs further investigation, as does also the question of what effect pressure on the vagus may have on the condition of the lung.

In considering the effect of pressure on the heart and the great vessels, we may practically disregard the left ventricle and the aorta, for it is from the left ventricle and through the blood in the aorta that the pressure is carried to the periphery of the aneurismal sac. We cannot expect this pressure to be greater than its source, hence it cannot compress the left ventricle or aorta. An aneurism arising at the root of the aorta may, however, by pressure distort either the aortic or pulmonary valve. In either case we get regurgitation into the corresponding ventricle as the chief factor in causing cardiac failure and death. Compression or obliteration of the innominate or left carotid or left subclavian arteries may occur. This leads to impaired nutrition of the parts supplied by these arteries, but the collateral circulation is so good that a fatal result is hardly to be expected from this cause alone, unless all three were occluded together, of which occurrence I have found no reported case. The supply of blood to the brain is, of course, the most

important item in considering obstruction of these arteries. Of the other branches of the general arterial system we need consider only the bronchial arteries—the nutrient arteries of the lung. Obliteration of one of these will cause gangrene of the part of the lung supplied by it, with fatal termination. This is a less frequent occurrence than was at one time supposed, as the bloodvessels of the roots of the lung are so situated that they are generally protected by the resistance of the bronchi.

Returning to the heart, we have to consider pressure upon the auricles, the conus arteriosus of the right ventricle, the pulmonary artery, and the pulmonary veins. In these cases we are dealing with obstruction of the circulation, essentially the same in its results as is found in valvular disease. Death may result in the same way as from broken compensation. Pressure on the vena cava superior is by no means uncommon. We get venous stasis of the upper part of the chest, the head, and the upper extremities. Collateral circulation may lessen this by furnishing a channel to the vena cava inferior, by anastomosis with its branches, or to the vena azygos if the pressure be above the point of entrance of that vessel into the vena cava superior. Here, again, the brain is the important organ to consider, and death by coma from the obstructed venous circulation is reported. Œdema of the glottis has also been reported[12] as a cause of death from impeded venous circulation.

Case III. is an illustration of death caused by pressure interfering with the circulation. The point of application was the pulmonary artery.

Male, aged forty-two years, painter, came to the out-patient department of the Boston City Hospital in the latter part of September, 1900. He had diphtheria eleven years ago. Otherwise he had been in good health, except for syphilis ten years before. He had used alcoholic drinks and tobacco to excess.

His present illness began about one year ago. He began to get short of breath on exertion, and also had vertigo and palpitation. For some months he had had pain at times in the præcordial region, radiating into the shoulders. Of late the pain had been worse and more frequent, and he complained of increasing dyspnœa and weakness.

On physical examination the cardiac area of dulness was practically normal. At the base of the heart was to be heard a peculiar murmur. It was loud and harsh in quality. Its point of maximum intensity was to the left of the sternum just above the pulmonic area. In rhythm it began just after the first sound—that is, it was a somewhat late systolic murmur—and continued just a trifle after the second cardiac sound; yet it was clearly all one murmur, and not a systolic running into a diastolic murmur. There was also a loud, ringing, markedly accentuated second pulmonic sound.

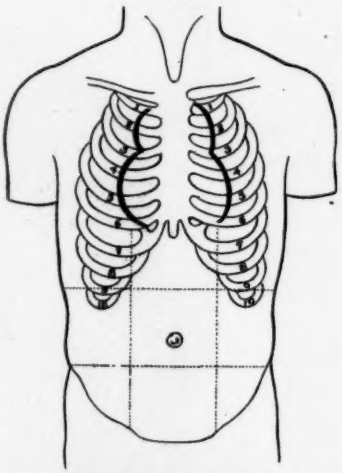
This peculiar combination is characteristic of a persistent ductus arteriosus. But it seemed highly improbable that such a case should live to this age without a longer history of cardiac trouble. The only other explanation which seemed at all likely was the existence of an

aneurism which compressed the pulmonary artery, and thus raised the tension in the pulmonary circulation. There was, however, no dulness, no pulsation, no tracheal tug, no hoarseness, no inequality of pupils, or any other physical sign to confirm the suspicion of aneurism of the arch of the aorta. He was asked to return for X-ray examination, but did not return till December 7th, when he was admitted to the hospital.

He was now much worse. He was weaker, had much more pain, more dyspnœa, had been unable to do any work for three weeks, and had had orthopnœa for two weeks. He had also become hoarse at these times. He was pale and had a slight cyanotic hue. The pulse was 80, regular, and of fair volume and strength. There was no perceptible difference in the two wrists. A distinct tracheal tug could now be felt.

There was now a slight bulging of the chest just to the left of the sternum, from the second interspace nearly to the clavicle. Over this area a slight pulsation could be seen; palpation revealed a distinct, coarse, rough thrill; percussion showed marked dulness; and on auscultation a rough, coarse, rumbling systolic murmur was heard. The second pulmonic sound was still somewhat accentuated, but was not as loud or clear-cut as before. Moreover, it was followed by a diastolic murmur, with its maximum intensity in the pulmonic area, transmitted downward and diagonally across the sternum to the right — following the location of the right ventricle and not the left.

FIG. 2.



Fluoroscopic examination. Sacculated aneurism of the ascending arch, growing forward and to the left, and compressing the pulmonary artery.

This murmur seemed to me to indicate pulmonary regurgitation. It was now perfectly evident that we had an aneurism pressing front to the chest wall to the left of the sternum. This had in September been too small to be detected, but so situated as to press on the pulmonary artery and greatly increase the resistance in the pulmonary circuit. This obstruction had now increased so much as to cause regurgitation through the pulmonary valve.

As a matter of interest an X-ray examination was made in this case also. It showed the heart slightly enlarged to the left, but markedly enlarged to the right. Above the base of the heart was an abnormally broad shadow, extending especially to the left of the sternum and very closely resembling the shadow of the aneurism of the descending arch, as shown in Fig. 1. The outline is shown in Fig. 2.

This patient gradually failed, and died December 17, 1900. Toward the end the attacks of dyspnœa were more frequent and severe. He had to be propped up in an upright sitting position for the last forty-

eight hours of life. During the last few hours he developed pulmonary œdema and his pulse gradually failed.

The autopsy showed a sacculated aneurism of the aorta compressing the pulmonary artery; hypertrophy and dilatation of the right ventricle; hypostatic congestion of the lower lobes of the lung, and atelectasis of an area of left upper lobe of lung, which was compressed by the aneurism; chronic passive congestion of liver, spleen, intestines, and kidneys; fatty degeneration of the heart and kidneys.

On removing the sternum the mediastinum presented a roughly spherical mass about 10 cm. in diameter, projecting forward and to the

FIG. 3.

Interior of left ventricle and aorta, showing opening into aneurismal sac just above the cusps of the aortic valve.

left. The mass is continuous with the heart and vessels of the base, and lies anterior and to the left of them. It projects 8.5 cm. to the left of the median line at about the level of the second rib. On dissection the mass is found to be an aneurismal sac 11 x 9 x 7.5 cm., which communicates with the aorta by an oval opening 2.75 x 2 cm. in diameter. This opening is situated in the anterior wall of the first portion of the aorta 3 mm. above the upper margin of the aortic cusps. The opening is on the left anterior aspect, above the junction of the anterior and the left posterior cusps of the valve. The edges of the opening are smooth and slightly raised. (See Fig. 3.)

Inside the sac a rounded mass of firm, organized clot fills the cavity to within ½ cm. of the opening. This mass becomes firmly adherent to the inner wall of the aneurismal sac at ½ to 1 cm. from the edge of the opening, except at the posterior side of the opening. Here a flattened pocket extends 3 cm. in depth between the rounded sac on the outside and the rounded mass of clot on the inside. The portion of this pocket farthest from the aortic opening is directly opposite the sinus of the right anterior cusp of the pulmonary valve. It is evident that this pocket is so situated as to have caught the greatest force of the blood

FIG. 4.

Aneurismal sac laid open, showing layered clot within.

current entering the sac, and this fact has prevented the formation of blood clot at this point. The cause of the peculiar murmur heard in this case was the eddying blood current in this pocket of the aneurismal sac.

Elsewhere the whole aneurismal sac is filled with firm clot. An incision from the anterior surface of the tumor shows a firm, fibrinous, dark red, layered clot, everywhere adherent to the inside of the sac. (See Fig. 4.) This aneurism not only had not ruptured, but it presented that condition, filled with firm clot, which is called "cured."

The general direction of the growth of the aneurism has been to the left and forward. It has also extended upward, as two-thirds of the mass is above the opening from the aorta. Still more important is the growth of the tumor a short distance backward, crowding in between the aorta and the pulmonary artery, so that where the transverse arch should cross close above the pulmonary artery it is separated from it by an aneurismal mass 5 cm. in thickness. The arch of the aorta has been apparently pushed upward, backward, and to the right to accommodate this growth; but there has been little, if any, diminution of the lumen of the artery.

FIG. 5.

Interior of right ventricle and pulmonary artery. Cusp of pulmonary valve stretched taut over bulging surface of aneurism.

In the pulmonary artery, however, a different condition obtains. There has been marked flattening from the encroachment of the tumor upon its lumen. The tumor presents as an irregular rounded mass, projecting downward and to the left into the pulmonary artery and the upper part of the right ventricle. (See Fig. 5.) The right anterior cusp of the pulmonary valve is stretched taut over the rounded surface of the tumor. The tumor projects into the right ventricle just below this valve, and the distance over its rounded surface is 4 cm. from the right ventricular wall to the upper edge of this cusp, and from this point to the end of the projecting mass in the pulmonary artery is 7 cm. The tumor mass as felt from the right ventricle or pulmonary artery is firm

everywhere except over the shallow pocket of the aneurismal sac already mentioned. This pocket lies behind the right anterior cusp of the pulmonary valve and part of the pulmonary artery just above it. Though yielding in the post-mortem specimen, it must have been firmly distended by the pressure of the aortic blood during life. The right anterior cusp of the valve must have lain flat over the surface, useless as far as its function was concerned.

The right ventricle was very much dilated, and the wall was hypertrophied to about four times its normal thickness. Death came from failure of the right ventricle, owing to the obstruction in the pulmonary artery and the regurgitation through the crippled pulmonary valve.

Pressure on the trachea or the primary bronchi is a frequent cause of death in aneurism of the aortic arch. This may act directly or indirectly. When it acts directly, death comes by suffocation, and the pressure is usually on the trachea just above its bifurcation. Death from this cause is rarely sudden; it is generally by slow asphyxiation, and the suffering is terrible. Sometimes the attack of asphyxiation is rather rapid in course, and unconsciousness ensues before there is much suffering, as in the following case:

Case IV.—Male, aged thirty-nine years, laborer, entered the Boston City Hospital, December 6, 1900. Family history was negative. He had had typhoid fever when young, and remembered no serious illness since then. At twenty-seven he had syphilis. Used alcohol moderately, tobacco to some excess.

His present illness began in August, 1900, four months before, when he began to have occasional spells of difficult breathing. He felt an attack coming on, would then sit down, and soon lost consciousness. If severe, the attack lasted about one and one-half hour. The attacks were followed by great prostration. The attacks came on rather suddenly after exertion or excitement. He had one just before entrance to the hospital; another five days before, and another five weeks before that. The intervals between the attacks had been variable. In the attacks breathing was extremely difficult, and he got blue in the face.

In the intervals between attacks he had pain and discomfort in the præcordia. There was some cough, but little expectoration. The voice had been husky since September.

Physical examination showed the pupils equal, the left vocal cord immovable, and the radial pulse equal and of good volume and tension. Tracheal tug was present. There was no œdema. Examination of lungs was negative. Upper border of cardiac dulness was at the third rib. The right border was one and one-half inches to the right, and the left border five and one-half inches to the left of the median line. Apex-beat was in the fifth interspace. A soft, systolic murmur was heard at the base, loudest to the left of the sternum. The second pulmonic sound was accentuated. Physical examination was otherwise negative.

Up to January 21, 1901, a period of about six weeks, he had three attacks—all of them severe. The dyspnœa was especially marked on expiration. He became cyanotic and lost consciousness. He recovered from these attacks, but was exhausted and weak for hours afterward.

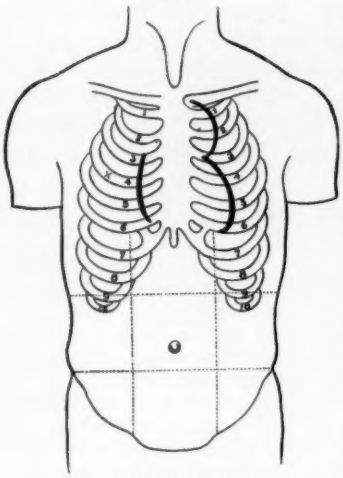
The hoarseness had continued, but varied in degree. At times he had a metallic, barking cough. There was dyspnœa on any marked exertion. He was able to be up and about the ward most of the time.

An X-ray examination had been made on December 21, 1900, and showed the following outline. (See Fig. 6.)

From January 21st to April 18th he had five attacks at varying intervals. On May 3d he had an attack. On May 5th and 6th he had four attacks. On the night of May 7th he had an attack during which he died.

The autopsy showed the following: sacculated aneurism of the transverse arch of aorta, and dilatation of the ascending arch; obstruction of trachea by pressure of aneurism; distention of lungs; chronic healed tuberculosis of lung; arterio-sclerosis; and hypertrophy of heart.

FIG. 6.



Fluoroscopic Examination. Sacculated aneurism of transverse arch, pressing on trachea.

The heart was somewhat enlarged, weighing 480 grammes, the principal change being in the left ventricle. The muscular tissue was firm and dark red, but showed some fatty degeneration on microscopical examination. The aorta is apparently dilated, and a mass about the size of a lemon lay below the clavicular notch, extending slightly into the left pleural cavity. This was a sacculated aneurism of the transverse arch of the aorta, which had not ruptured.

The aortic orifice measured 8.5 cm. in circumference, and the ascending arch was somewhat dilated, measuring from 9 to 10.5 cm. in circumference. At a point 9.5 cm. from the aortic valve there is a rounded border forming part of the opening of an aneurismal sac. This opening measured 8 by 9 cm. in diameter. This sac arose almost entirely from the posterior and inferior part of the transverse arch, while the innominate, left carotid, and subclavian arteries arose in front from the apparently normal aortic wall, and were practically normal. The sac was hemispherical in shape, and the greatest circumference of it and the dilated aortic wall was 22 cm.

The aneurism lay directly anterior to the trachea just above its bifurcation. The trachea was flattened and curved back by the pressure, and its lumen was encroached upon from the anterior and left side by the bulging wall of the aneurismal sac. Even with the sac empty the lumen of the trachea was only one-third of its normal size at this point.

The lungs were very much distended with air. When the sternum was removed they did not collapse, but bulged forward, nearly covering the heart. On cutting the bronchi the lungs collapsed to less than one-half their former size from the escape of air. There was an area of

healed tuberculous tissue occupying about one-third of the right upper lobe. Elsewhere the lung tissue was normal. The bronchi were normal. The bronchial lymph nodes were normal.

It was clear that the distention of the lungs was due entirely to the pressure of the aneurism on the trachea. The pressure of the distended sac must have been sufficient to entirely prevent the exit of air, and the patient died of suffocation. It is not difficult to understand how the air passed by this obstruction on inspiration, but could not escape again. With the descent of the diaphragm on inspiration the heart and anterior part of the arch are drawn downward. This drew the aneurism away from the trachea enough to allow the entrance of air, but when this traction ceased at the end of inspiration the pressure was at once resumed, and the aneurism acted like a ball valve to prevent the exit of the air. The resulting dyspnœa caused more forcible inspiratory efforts, the entrance of more and more air which could not escape, and the extreme distention of the lungs resulted. With the complete distention of the lung no more air would enter, and respiration was brought to a standstill, and death ensued from suffocation.

Compression of one bronchus is not so likely to cause death from suffocation. It is rarely so sudden that the necessary supply of air cannot be obtained through the other lung. Death is more likely to come through secondary changes in the lung. How far these secondary changes may be the result of pressure upon the bloodvessels or nerves at the root of the lung has been a matter of dispute, and I do not think we are yet prepared to decide the question definitely. It is, however, certain that the mere mechanical occlusion of the bronchus may result in extensive and fatal changes in the lung tissue. This is due to the retention of secretions and the invasion of micro-organisms. Collapse and consolidation of lung tissue, a purulent bronchitis extending to the smaller divisions, and then to the lung tissue, and causing multiple foci of suppuration, and gangrene, are the manifestations of pulmonary disintegration which follow compression of the bronchus. Death in this case comes as an indirect result, not as the direct result of the pressure of the aneurism. Irvine[10] has reported a case of this sort, and has called attention to the subject. Compression of one primary bronchus may, however, produce collapse and atelectasis of the corresponding lung without further secondary results, as is shown in Case V.

Finally, in considering the effects of pressure on the pulmonary tissue, we must note the occurrence of cases where the aneurism is so situated that it does not press on the main air passages, but upon the lung tissue itself. Here we get primarily a compression of the lung tissue, and may get secondary inflammatory processes in the tissue thus affected.

Pressure on the œsophagus may become so extreme as to prevent the passage of food and cause death by inanition. In less degree it may be

a more or less important factor in causing insufficient ingestion of food and imperfect nutrition, thus rendering the patient an easier victim to intercurrent disease of any kind. Compression of the thoracic duct may cause serious impairment of nutrition with similar relations to the fatal termination, but this is extremely rare.

The following case illustrates death by starvation from the pressure of an aneurism on the œsophagus.

Case V. Female, aged fifty-seven years, seamstress, had always enjoyed good health before the present illness. In the fall of 1893 she first noticed a pain in the lower left axillary region, which also shot into the back and up to the left shoulder. This continued at intervals.

In the fall of 1895 she began to have partial aphonia and difficulty in swallowing. After a time solids could not be swallowed at all, but she could swallow liquids. By the last of January, 1896, a hacking cough had set in. Sibilant and sonorous râles were heard over both lungs. By March there was considerable expectoration, which was occasionally streaked with blood. By April dyspnœa was caused by even slight exertion. She was very weak. She had been losing flesh since fall, and was now greatly emaciated. Even liquids were now vomited or regurgitated, and nutritive enemata were resorted to after April 5th. She failed gradually, and died quietly on June 2, 1896. No new symptoms had developed, and all the old ones had persisted.

During the latter part of her illness it was noticed that the respiration was very faint on the right side of the chest. Tuberculosis of the lung was thought of, but the diagnosis would hardly account for all the symptoms, and no tubercle bacilli could be found in the sputum. The probable diagnosis was thought to be a malignant tumor of the œsophagus. This would account for the obstruction of the œsophagus, the wasting, and the pain, and, by extension and pressure on the left recurrent laryngeal nerve and right bronchus would account for the aphonia and diminished respiration of the right lung. This clinical diagnosis represented very closely the autopsy findings, except that the tumor was an aneurism of the aorta instead of a malignant tumor of the œsophagus.

The autopsy showed a sacculated aneurism of the thoracic aorta; atheroma of the aorta; pressure of aneurism on right primary bronchus and on œsophagus; atelectasis of right lung; bronchitis of left lung, and all other organs normal.

The aneurism was a sacculated one, about three and one-half inches in diameter, arising from the upper part of the descending thoracic aorta. It had grown forward and to the right, crowding in between the œsophagus behind and the air passages in front. The œsophagus was compressed flat against the vertebral column. The right primary bronchus was so completely occluded by the pressure toward the front that the right lung had collapsed and was in a state of atelectasis, but no inflammatory change had resulted. The fourth to sixth dorsal vertebræ (inclusive) were eroded on their right side from the pressure of the sac. The aneurism was partly filled with lamellated clot, and had not ruptured. The thoracic duct could not be found. The anatomical relations of the aneurism, the absence of other pathological cause, and the very extreme emaciation showed that death came from starvation, of which the aneurism was the immediate cause.

I wish to acknowledge my indebtedness to Drs. Withington, Williams, and Sears, of the staff of the Boston City Hospital, for permission to report the progress of Cases I., III., and IV. while under their charge in the wards; to the pathological department of the hospital for the excellent reports of autopsies; to Dr. H. C. Williams, of Boston, for the history of Case I., and to Drs. S. H. Ayer and W. H. Prescott for the clinical history and autopsy report of Case V.

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THE ASSOCIATION OF PULMONARY TUBERCULOSIS WITH BOTH PRIMARY AND SECONDARY ENDOCARDITIS, AND THE EFFECT OF VALVULAR DISEASE UPON LUNG TUBERCULOSIS.*

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THE subject of the relationship existing between diseases of the endocardium and pulmonary tuberculosis is admittedly but imperfectly understood. The cases of endocarditis met with in tuberculosis of the lungs are clearly divisible into several classes, as follows: 1. Those due to the presence of tubercle bacilli within the heart—*endocarditis tuberculosa*. 2. Those that are secondary to tuberculosis or merely intercurrent, and caused by various organisms other than the tubercle bacillus. 3. Various forms of valvular heart disease that precede the tuberculous infection of the lung and are due to rheumatism and other etiological agencies.

A distinction between these different varieties has not, as a rule, been

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drawn by writers, hence the important practical question of the relative frequency of the occurrence of each in association with tuberculosis of the lungs has not been definitely settled. These varied classes of cases demand separate study if we would obtain positive and practical information on the general subject. Thus the mere detection of the co-existence of pulmonary tuberculosis with acute or chronic endocarditis at necropsy, without knowing anything of the special causation of the cardiac lesions, their nature, or whether a tuberculous lesion of the endocardium exists or not, is of no scientific and little if any practical value.

Although we are sadly in need of reliable and more elaborate statistics, it is my purpose, in connection with a general consideration of the practical phases of the subject, to tabulate all recorded cases according to the classification given above, satisfied that a collective investigation and a discriminating study made on these lines will lead to greater unanimity in establishing conclusions.

1. *Cases of endocarditis due to the tubercle bacilli.* In 1806 Corvisart[1] pointed out the existence of little tubercles situated on the mitral valve in an individual afflicted with tuberculosis of the lungs and the pericardium, coupled with caries of the ribs and vertebræ. It remained, however, for Wagner[2] to give the first description of tuberculosis of the endocardium. In 1886 Lancereaux observed a case of endopericarditis of a tuberculous nature. From the date of the observations and more complete studies of Letulle (1874) and of Perroud (1875) a proper classification of the lesions of tuberculous endocarditis was attempted. Later, Kundrat also found the tubercle bacillus in the endocardium, and the observation has been abundantly confirmed by others. It is interesting to note that while Ponfick, Weigert, Sticker, and others have found the tubercle bacilli commonly present in the blood, true tuberculous endocarditis is a truly rare condition. Perroud long since pointed out that small aortic vegetations sometimes occur in children dying of general miliary tuberculosis. Indeed, involvement of the endocardium is most apt to occur in acute miliary tuberculosis; but we are not especially concerned here with this clinical variety of tuberculosis. I shall also endeavor to exclude the cases of tuberculosis of the endocardium resulting from direct extension from adjacent parts, as in tubercular invasion of the mediastinal glands.

The fact is to be emphasized at the outset that the endocardium, like the interior of the bloodvessels, is to an unusual degree resistant to the tubercle bacillus. Infection of the endocardium most probably takes place through the blood-supply to the heart structures, or possibly through that which finds its way into the cavities from the endocardium, and not the blood circulating within the chambers of the organ. It is known that when the arteries in a tuberculous area become involved the adventitia, and not the intima, is first affected. The endocardium

then fulfils a decidedly protective function so far as invasion of its structure by tubercle bacilli is concerned. In this connection it is interesting to note that, as regards frequency of occurrence, Bollinger gives cardiac tuberculosis fourteenth place among the viscera. Here it should be recollected also that the pericardium and the myocardium are oftener the seat of tuberculosis than the endocardium.

Teissier,[3] in an extensive monograph on the subject of tuberculous endocarditis, states that during a period of three years he examined minutely the hearts of all cases of tuberculosis—chronic, acute, and miliary—and was unable to find a single example of true tuberculous endocarditis. He cites Potain's case of tuberculous myocarditis and Landouzy's numerous cases of infantile tuberculosis in which the endocardium was uniformly free from tuberculous lesions.

The figures in the accompanying Table I. (cited mostly from Teissier's monograph[4]) are derived from the earlier literature on the subject, and they serve to emphasize the then prevailing view that tuberculous endocarditis in the course of pulmonary tuberculosis practically never occurs.

TABLE I.

Name.	Total number of necropsies.	Cases of tuberculous endocarditis.	Percentage of cases.
Sänger	469	3	0.64
Willig	845	None.	
Reisner	152	"	
Chambers	566	"	
Total number of cases	2032	3	0.148

The figures gleaned from more recent available sources, however, and tabulated below (Table II.) convey a different idea as to the frequency of the occurrence of acute endocarditis in the course of pulmonary tuberculosis, although it is unquestioned that a close analysis of the cases recorded shows the great majority of them to have been due to excitants other than the tubercle bacilli. In cases in which the lesions were of the ulcerative variety, at all events secondary pyogenic infection probably existed.

TABLE II.

Name.	Number autopsies in tuberculosis.	Recent endocarditis.	Per cent.	Remarks.
Osler[5]	216	12	5.5	Mitral valves 8, aortic 3, a. and m. 1.
Bernard Schultz[6]	6937	67	0.966	In 42 other cases the endocarditis was primary.
Percy Kidd[7]	500	6	1.2	In 21 other cases the endocarditis was probably due to other causes.
Teissier[8]	100	32	32	In 12 the tubercle bacilli were present on the valves (cited cases).

Additional statistics are to be found in the literature, but they bear upon the general subject of valvular disease associated with phthisis, or indicate no attempt to discern recent tuberculous endocarditis from other forms of endocarditis, hence they are not available for my present purpose. Thus Krygen[9] autopsied 1100 cases of tuberculosis, and in 10 found valvular lesions associated.

H. Walsham[10] met with 21 cases of endocarditis in 130 sections of cases of tuberculosis, but no mention is made of the particular varieties of endocarditis encountered.

As before mentioned, it is not improbable that the cases of recent endocarditis included in the above table were due to causes other than tuberculosis, for the reason that these cannot readily be excluded. Osler expresses doubt concerning the tuberculous nature of his own cases as well as of those reported by others. As opposed to this view it must be recollected that that eminent authority has favored us with the avowed opinion that more than one-half of the cases of endocarditis associated with chronic phthisis, even including the sclerotic variety, are secondary to the pulmonary lesions. This aspect of my subject is of great practical importance, and there is need for more extended study and investigation.

Clinically, the cases of tuberculous endocarditis are extremely difficult of recognition. The history of the case, however, may be of diagnostic significance. If it can be shown that the cardiac affection developed subsequent to undoubted pulmonary tuberculosis, and if rheumatic and other forms of infectious endocarditis can be eliminated, and especially if there have been neither previous arterio-sclerosis nor fibroid degeneration of the viscera, then a reasonably certain diagnosis of tuberculous endocarditis, given the usual signs and symptoms, can be made. The condition may also be overshadowed by the severer symptoms of the closing stage of pulmonary tuberculosis.

The clinical association of pulmonary tuberculosis with recent endocarditis is illustrated by a single case out of a total of ninety-two cases of tuberculosis of which I have satisfactory records.

A. T., aged thirty years; occupation, clerk; without vicious habits. Predisposition to tuberculosis was an inheritance from both sides of the ancestry. No previous rheumatism, although the patient had had scarlet fever and measles during childhood. Pulmonary tuberculosis developed at twenty-four years of age and pursued a comparatively rapid course, terminating fatally at the end of eighteen months amid the signs of extensive cavity formation. February 1, 1894, six weeks prior to death, a distinct soft bruit made its appearance at the apex of the heart; the area of transmission, however, was quite limited. The impulse was somewhat diffuse, stronger at first, then growing feeble, and the cardiac dulness extended to the left vertical mammary line. The second sounds were not accentuated, and there were no associated murmurs.

Although no necropsy was permitted after death, the case was regarded as one of probable tuberculous endocarditis after the exclusion of other causes to explain the condition.

Fenwick, Traube, and many others have made clinical reports of instances of tuberculous endocarditis; but I have found that they lack definiteness as well as all bacteriological evidence. It would appear from my studies that almost all of the rare instances of true tuberculosis of the endocardium are associated with other forms of cardiac tuberculosis, especially the pericarditic. In this connection I may mention the twenty-eight observations of tuberculosis of the myocardium (cited by Teissier) in which no lesions of any portion of the endocardium were found.

But though tuberculous endocarditis is so extremely rare as to be almost non-existent, and hence of slight practical import, tuberculous sclerotic endocarditis is not uncommon, as demonstrated by pathological investigation. Lebert has noted three cases of the sort. It is extremely difficult to eliminate the influence of antecedent rheumatism and other infections, as well as that of gout, syphilis, chronic rheumatism, occupation, and other recognized causes of sclerotic endocarditis. This can, however, be done, as a rule, in the cases occurring in young subjects; and Teissier found that sclerosis occurs in 40 per cent. of the instances, even after excluding other etiological factors, as rheumatism, etc. This sclerosis of the endocardium is probably ascribable to an intense and oftentimes protracted intoxication of the economy. Neither tubercle bacilli nor tuberculous changes have been detected in these thickened areas on microscopical examination. Teissier also affirms that progressive narrowing of the mitral orifice is due to tuberculosis. Sansom,[11] while not in entire accord with Teissier, states that there is some relation between mitral stenosis and pulmonary tuberculosis. Drs. Harris and Beale[12] met three cases of mitral stenosis in a series of 133 post-mortem examinations on cases of tuberculosis. On the other hand, Walsham remarks, "I suppose all who have made many post-mortem examinations on cases of pulmonary tuberculosis will admit that the endocardium is frequently found thickened in places, especially in the left ventricle in the neighborhood of the aortic segment of the mitral valve, but never to the extent of producing a narrowing of the mitral orifice." He concludes: "I think that mitral stenosis with pulmonary tuberculosis is so rare a condition that the one cannot be regarded as the cause of the other, although not antagonistic to each other."

Percy Kidd, in speaking of his cases of combined heart disease and phthisis, previously cited, observes: "It is uncertain whether endocarditis or phthisis was established first in more than half the total number of cases, though in the majority of these it is fair to assume that tuberculosis was the primary disease." The occurrence of rheumatism was

noted in twelve of Kidd's cases, "and it is remarkable that the rheumatic cases almost exactly corresponded to those in which old endocarditis and dilatation or hypertrophy were found. In the non-rheumatic cases it is difficult to account for the development of granulations on the cardiac valves in the course of phthisis. Whether or not there may be any etiological connection between the tuberculous process and endocarditis must remain at present uncertain." He did not succeed in discovering tubercle bacilli or any tuberculous structure in the granulations in his cases. Out of a total of forty-two cases of so-called tuberculous endocarditis collected by Teissier, only twelve were supported by bacteriological evidence. It has been questioned by some authors whether the detection of tubercle bacilli in the valvular vegetations warrants us in pronouncing positively in favor of tuberculous endocarditis, since the granulations may simply afford a resting-place for the organism.

Leyden,[13] in writing on the subject of tuberculous endocarditis, alludes to four cases of acute endocarditis in his own experience in which tubercle bacilli were discovered in the vegetations. In his opinion the acute endocarditis was not due to the tubercle bacillus, their presence on the segments notwithstanding. In one of my cases the typical physical signs of both mitral stenosis and insufficiency were associated with the unequivocal signs and symptoms of pulmonary tuberculosis, including the presence of tubercle bacilli in the sputum, from the date of admission to the Medico-Chirurgical Hospital. The patient was a male, aged twenty-seven years, and gave no history of antecedent rheumatism. Such cases, and they are not very uncommon, cannot be reliably classified. To say that the chronic endocarditis was secondary to the lung infection and due to the same cause, would be a purely prophetic utterance.

2. *Cases of recent endocarditis and chronic endocarditis that are secondary to tuberculosis or merely intercurrent, and are caused by various non-tuberculous infections and other agencies.* That such cases do occur, due to invasion by the streptococcus, staphylococcus, etc., is beyond peradventure, and most of the instances that are ascribed to tuberculous infection have in reality a different etiology. As stated elsewhere, not less than one-half of the cases of chronic valvular disease are caused by rheumatism, and more than one-half of the total number occur between twenty and thirty years of age, a period of life during which pulmonary tuberculosis is also quite frequent. In the vast majority of the cases, then, chronic endocarditis originates in primary acute rheumatic endocarditis, which almost invariably terminates in the former affection. Acute rheumatism, however, rarely complicates chronic phthisis, according to my observation. Indeed, it may be affirmed that both the gouty and the rheumatic diathesis are largely exempt from pulmonary tubercu-

losis. In Pollock's[14] experience, "no history of rheumatism is obtainable in a considerable proportion of the cases." Percy Kidd, however, noted rheumatism in twelve of his twenty-seven cases of endocarditis associated with pulmonary tuberculosis.

Other causes, such as syphilis, malaria, alcohol, lead, and even occupation may rarely produce intercurrent chronic endocarditis in the course of pulmonary tuberculosis. My notes probably furnish a case in point:

A young man, colored, aged thirty years, with atheromatous vessels due to acquired syphilis, contracted tuberculosis for which he applied to the out-patient department of the Episcopal Hospital of Philadelphia. Six months later he presented himself a second time with the signs of aortic regurgitation, with moderate left ventricle hypertrophy. The case progressed from bad to worse, with respect both to the tuberculosis and the valve complaint, and finally his visits ceased and the case was lost sight of.

Although developing after the onset of the pulmonary condition, the aortic regurgitation was, in view of the vascular changes and the previous history, believed to have been occasioned by the syphilitic rather than by the tubercular infection. Both acute and chronic endocarditis may be secondary in the course of pulmonary tuberculosis.

3. *Forms of chronic valvulitis that are primary and precede the tuberculous infection of the lung, and are ascribable to rheumatism and other recognized causes.* This is the most important group of cases to be considered. This class has manifestly a non-tuberculous etiology, and it is believed that the cases exert in the main a contrary effect according as they affect the right or the left side of the heart. Hence, this division of the subject falls naturally under two heads: 1, The influence of left-sided heart affections (mitral stenosis, mitral regurgitation, aortic stenosis, aortic regurgitation) upon the development and clinical course of pulmonary tuberculosis; and 2, the influence of right-sided chronic valvulitis, more especially stenosis of the pulmonary ostium upon the etiology and clinical course of chronic phthisis.

The subject has occasionally engaged the attention of clinicians from the time of Laennec down to the present, and for a detailed statement of the views of the older writers I must refer my auditors to the excellent article of J. E. Graham,[15] of Toronto, read before the Canadian Medical Association at Montreal, 1896.

Rokitansky's law, that "Persons laboring under enlargement (dilatation, hypertrophy, and their complications) of the heart, whether primary or superinduced by mechanical obstruction at its orifices, do not contract tuberculosis," is untenable. Such noted authorities as Niemeyer, Traube, Ruehle, and Pollock, while not holding to an absolute antagonism, yet with one accord maintain by their writings that a

decided protection is offered against pulmonary tuberculosis by the occurrence of valvular heart disease.

Graham collected some statistics bearing upon the effects of valvular heart disease on the existence of pulmonary tuberculosis previous to the date of publication of his paper, 1896, and I have tabulated these below (see Table III.). Unfortunately, the figures may include cases in which it cannot be positively stated that the cardiac affection was primary and the tuberculosis secondary.

By this table the broad fact that pulmonary tuberculosis occurs less commonly secondary to valvular heart disease than as a primary disease under ordinary circumstances receives some support. But, as previously stated, this counteracting influence is less potent than formerly supposed, and, as shown in Table IV., is a somewhat variable quantity depending upon the particular orifice implicated. The subjoined Table III. sets forth the influence of valvular affections as a whole upon the development and course of pulmonary tuberculosis:

TABLE III.

Name.	Number of cases of organic valvular disease.	Ante-mortem.	Post-mortem.	Number of cases of tuberculosis.	Percentage of cases of tuberculosis.
Bernard Schultz[16] . .	484	.....	484	22	4.5
Alfred Eyman[17] . .	4138	4138	.....	69	1.7
Frommolt[18] . . .	276	.....	276	21	8
Germain (see Hanot)[19] .	277	277	.....	22	8

In the majority of the cases in which the special lesions were given, combined forms were observed, hence it was not possible to collect a sufficient number of single lesions to establish the relative frequency of the association of pulmonary tuberculosis with the separate recognized valvular affections. On comparing the percentages given above with the well-known ratio of cases of pulmonary tuberculosis to the general population, one is not struck by the extreme rarity of this disease secondary to simple valvular heart lesions. If the cases that were autopsied (760) are considered alone, the percentage of instances complicated with phthisis rises to 16.1, while the clinical cases, 4405 in number, give a percentage of only 1.8. To account for these contrasted results in the ante-mortem and post-mortem findings, one is forced to conclude that in most cases of valvular disease presenting pulmonary tuberculosis as a complication, the latter goes unrecognized during life. Moreover, the tuberculosis is often latent.

Bollinger's[20] series of necropsies on subjects under fifteen years of age show that among 218 cases of pulmonary tuberculosis 68 were of this character. The association of phthisis with primary valvular

heart disease is probably more common than the usual clinical findings have thus far indicated.

In answer to a series of questions published in sundry periodicals as to the association of valve lesions with pulmonary tuberculosis, a few responses have been received, and these coupled with my own personal records are here tabulated:

TABLE IV.

Name of reporter.	No. of cases of pulmonary tuberculosis.	Valve lesions.	Per cent.	Particular valve lesions.
Dr. L. B. Milliken	60	3	5	1 mitral stenosis. 2 mitral regurgitation.
Dr. E. W. Huhner..	36	1	2⅞	Cardiac hypertrophy.
Dr. A. Abrams	800	21	2⅝	1 pulmonary stenosis. 2 aortic stenosis. 3 mitral stenosis. 9 mitral regurgitation. 3 mitral stenosis and regurgitation combined. 3 unclassified.
Dr. J. M. Anders	94	6	6⅜	4 mitral regurgitation. 2 mitral stenosis.
Dr. E. Baldwin	1,200	9	¾	7 mitral regurgitation. 1 aortic regurgitation. 1 mitral stenosis.
Dr. A. Eyman[21]	27,653	80	0.288	Does not give individual lesions.
Dr. H. Walsham[22]	1,000	1	0.001	1 mitral stenosis. As to other lesions nothing is stated.
Total exclusive of Eyman's, Baldwin's and Walsham's cases	990	31	3.13	

These figures indicate that the observations upon cases of phthisis give a comparatively low percentage of instances in which this disease occurs in primary valvular lesions. I confidently believe that a different result would have been obtained by those observers who report the fewest cases of primary valvular heart disease if their attention had previously been especially directed to the association under discussion. This is confirmed by my own results as well as those of Dr. Milliken, who found three cases among sixty patients at Loomis Sanitarium, Liberty, New York, at one and the same time, after his attention had been drawn to the subject by my inquiries.

A clinical diagnosis of valvular heart disease must be made with due reserve and caution, there being murmurs at various orifices and apparent hypertrophies met in pulmonary tuberculosis that are not dependent upon valve lesions; but to enter upon a discussion of their nature and discrimination from the latter here would lead me too far. Oppositely, I have observed two cases of mitral stenosis in which slight consolidation of the lung above the heart existed, but the usual crucial tests did not reveal pulmonary tuberculosis. The diagnosis of cases of combined cardiac disease and tuberculosis of the lungs demands either the presence

of tubercle bacilli in the sputum or a positive tuberculin test in connection with clear evidence of valvular heart disease. Sansom has called attention to the fact that many cases of mitral stenosis show signs which can be easily mistaken for those of pulmonary tuberculosis.

The question naturally arises here, What is the explanation of the immunity offered by the lesions of the valves in the left heart against pulmonary tuberculosis? The responses that I have received from leading members of the medical profession in answer to this question, with few exceptions, offered the opinion that the preventive effect is ascribable in the main to pulmonary congestion. Albert Abrams, however, ascribes the antagonism to cardiac hypertrophy. Brehmer first ably championed this theory: he stoutly maintained that the disposition to and the cause of phthisis was a small, weak heart. Commenting upon a series of eighteen patients in whom valvular disease and pulmonary tuberculosis co-existed, Abrams pertinently remarks: "These cases thrived admirably as long as compensation was maintained, but when failure of compensation supervened the phthisical signs were fulminating. The pulmonary symptoms seemed to be held in abeyance during the continuance of compensation."

Says Graham: "It seems probable that the immunity from pulmonary tuberculosis existing in mitral disease is due to the following conditions: 1, The increased amount of blood in the lungs; 2, the greater expansion of the apices; 3, the increase of the involuntary muscular fibres of the bronchi." Graham affirms that among the changes observed in brown induration, the increase of connective tissue and the deposit of pigmentary matter are of no value in preventing tuberculosis. Concerning the fibroid induration, it is but reasonable to suppose that it plays a rôle of prime importance, seeing that it is by the formation of new connective tissue that healed tuberculosis is made possible.

There ensues more or less enlargement of the lungs in consequence of passive congestion in mitral disease, but it is an error to assume that the expansive power of the lungs is noticeably augmented, as can be readily shown by the use of the tape in cases of diseases of the mitral segments. Opposed to the view that pulmonary congestion *per se* is the all-important factor in preventing the development of pulmonary tuberculosis is the fact that hæmoptysis which diminishes the hyperæmia often affords considerable relief from symptoms and even arrests temporarily the march of the disease.

Again, respecting the rôle played by venosity of the blood, on which some writers, including Rokitansky, have laid stress as a factor in the supposed antagonism existing between phthisis and heart disease, it is to be particularly remarked that this condition is a conspicuous feature in congenital pulmonary stenosis which is more commonly associated with phthisis than mitral disease (*vide infra*).

It must be conceded that passive congestion in the lesser circulation or the local anatomical changes it produces (brown induration) or both seemingly account for the comparative immunity enjoyed from phthisis by those who have primary chronic valvular disease; this probably implies an increased supply of nutritious material to the lungs rather than that the tubercular virus is opposed directly by the venosity of the blood. It is a question of lung-nutrition.

Left-sided valvular heart disease exerts an influence upon the symptomatology of phthisis. In reviewing the history of cases, as well as from personal experience, I have been struck with the similarity between the general course of some of the cases that are combined with heart lesions and uncomplicated pulmonary tuberculosis. Some of the leading symptoms, however, show noticeable variations. Thus, from an examination of the notes of the cases gleaned from the literature (including also six of my own cases) a prominent initial symptom is not uncommonly hæmoptysis. Pollock observes that in the chronic phthisis associated with chronic valvulitis, hæmoptysis is more frequent and more severe than under other circumstances, and Balfour[23] also directs forcible attention to the same fact. I have records of six cases (see Table IV.), and in two of these hæmoptysis was the initial objective symptom for which relief was sought by the patients, and yet in both a physical examination revealed lung-involvement and the sputum-test the presence of tubercle bacilli directly after its occurrence.

The practical lesson to be learned from this bit of experience is that tuberculosis secondary to chronic valve lesions may remain latent for a longer or a shorter period, and is then apt to be announced by the supervention of blood-spitting. Once started, the hæmoptysis showed a greater tendency to recur in my cases than in non-cardiac cases, and in one instance the bleedings were so profuse, and that at a comparatively early period as to cause an almost fatal degree of shock. A change of air to the Adirondack region and the free use of digitalis produced a cure so far as the phthisis was concerned.

When not so copious as to be immediately serious, these hemorrhages have seemed in some instances to have been followed by an amelioration of the cough and expectoration and less commonly by an improved appetite and general condition.

The dyspnœa is more pronounced in cases of phthisis combined with valvular heart disease than in uncomplicated phthisis, and is of the cardiac type. Again, the temperature pursued a lower range in my combined cases than in phthisis occurring independently of cardiac disease.

There is perfect agreement among writers that valvular heart disease has a retarding influence upon the progress of chronic phthisis. According to my experience and an examination of the literature, however,

this is true in a more limited sense than is usually supposed. While the development of phthisis is delayed in many cases combined with heart disease, it must be recollected that " when the disease attacks an individual after middle life, or when from bad habits or bad hygienic surroundings there is a low state of vitality the process is often rapid " (Graham). Excessive and frequent bleedings may prove an element of danger, and they may shorten the duration of a certain number of the cases. Moreover, the lung condition tends to aggravate the cardiac affection, and the error too often committed, I find, of sending these patients to resorts that have an elevation of more than 2000 feet produces an unhappy effect upon the cardiac lesions, and may undoubtedly hurry a fatal termination. In briefly summing up the effect of the heart lesions upon the course of the phthisis it may be safely assumed that in the main there is a distinct retarding effect manifested under judicious handling of the cases. The major treatment, however, has reference to the cardiac element no less than to the phthisis.

Finally, it is only in cases in which valve lesions and the compensatory hypertrophy are proportional that a prognosis for unusual length of days can be safely ventured. When the harmonious balance is disturbed in the slightest degree an early fatal termination may be expected principally from the cardiac complaint. I append here brief notes of my own cases:

Case I.—C. C. R., aged seventeen years, at school, called first on March 4, 1899. One brother had had hæmoptysis. Patient had been complaining for a year of extreme nervousness and nocturnal emissions; also cough which for a long time had been unproductive, but later attended with expectoration containing tubercle bacilli.

Physical signs referable to lungs: weak vesicular murmur at the left apex with a few subcrepitant râles over bronchi and slight impairment of the percussion resonance. Heart: moderate hypertrophy of both ventricles, apical systolic murmur and accentuation of pulmonary artery second sound. August 15, 1901, patient apparently cured of the phthisis as the result of change of residence from the city to the country, forced feeding, and out-of-door life.

Case II.—J. A. C., aged twenty-eight years, occupation glass decorator, temperate habits, brought to me by Dr. O'Reilly. The entire family, save father and one brother, died of phthisis. No memory of previous rheumatism, but subject to frequent " colds." His illness started as a heavy " cold " in April, 1900; there were cough and expectoration, the latter becoming abundant, and later containing tubercle bacilli.

Physical Signs. Both apices gave the signs of decided infiltration, while the heart presented those of mitral regurgitation. This patient removed to Denver, where, as I learn from Dr. O'Reilly, he died within one year.

Case III.—A. L., aged eighteen years, male, at school. Family history negative. About three days prior to his first visit spat one-half pint of blood; this was followed by cough and a thick, yellowish expectoration. History of frequent attacks of hæmoptysis in the mean-

while. No sputum test was made by me, although from the well-marked physical signs referable to the left apex, no doubt would be entertained as to the correctness of the diagnosis of pulmonary tuberculosis. The heart presented the typical physical signs of mitral stenosis combined with regurgitation. The final outcome in this case could not be learned, but at the time of patient's second visit, about six months after his first, it was noted that the pulmonary lesions had made little progress, and the hæmoptysis, the patient declared, was followed by marked, though temporary relief from cough and expectoration.

Case IV.—C. A., aged thirty-seven years, married, female, first fell under my care November 16, 1900.

Family History. Maternal relations all died of phthisis, otherwise negative.

Previous History. Childish diseases; at ten years of age had articular rheumatism; and six years ago had second attack, lasting six weeks. Malaria at twenty-five years of age for three months or more. Her present illness began January 1, 1900, with slight cough and expectoration; three months later gave birth to a child, after which the cough grew slightly worse. In July (1900) had hæmoptysis; five weeks later had another seizure, losing about a pint of blood. One month later hemorrhages recurred at frequent intervals until her life was despaired of. Since then has had lighter ones. Has not lost much weight; occasional night-sweats. The physical signs at first visit: Lungs: Evidence of slight infiltration of left apex region, clear and distinct. Heart presented the signs of typical mitral regurgitation. Tubercle bacilli have been repeatedly found in the sputum. The diagnosis of combined mitral regurgitation and phthisis was readily made in view of the history of previous rheumatism, slight extent of the pulmonary lesions on the one hand, and the well-marked signs of mitral incompetency (including decided ventricular hypertrophy) on the other. Doubtless the heart changes were primary. The course of the lung complaint since the occurrence of childbirth has been found much the same as in the absence of associated heart lesions, compensation remaining apparently good notwithstanding.

Case V.—M. B., aged twenty-five years, married, occupation sewing machine operator; first came under observation September 3, 1898. No traces of any hereditary taint in the family, and his own health uniformly good until April, 1898, when the illness for which he consulted me began. He first developed a severe "cold," which improved from time to time, but never entirely disappeared. Three months previous to his first visit he expectorated blood-stained sputum. August 28th, had a recurrence of the hæmoptysis which lasted for three days. Since then has had a cough and slight expectoration, with occasional night-sweats, but thinks his bodily weight is maintained. The physical examination showed a paralytic thorax and the signs of commencing consolidation at the upper portion of the left lung. The heart presented the usual physical signs of mitral incompetency; the murmur, however, was more marked after muscular exercise. Compensation has been good. The progress of the phthisis was exceedingly slow and finally, perhaps, arrested. I have not been able to learn anything concerning his condition for the past year and a half.

Case VI.—J. C. O, aged twenty-four years, single, occupation farmer, was brought to me for examination by Drs. Robin and Dugler,

on May 9, 1899. The family history was negative. The patient had had the usual child's diseases, and in 1889 contracted the grip, with which he was afterward frequently afflicted. In September, 1896, had developed a cough, which remained until the date of his first visit; this was accompanied by free mucopurulent expectoration. There had been a falling off in weight of not less than fifty pounds. The physical signs of a moderate-sized cavity at the left apex were present, combined with those of mitral stenosis. The course in this case had been quite protracted until the signs of failing compensation developed, after which a fatal termination amid the signs and symptoms of advanced cardiac disease was not long delayed.

I have not observed a single case of aortic valve disease associated with phthisis since my attention was especially directed to the subject of this paper, some five years since, and it will have been observed that Table IV. furnishes a single case of both aortic stenosis and aortic regurgitation out of a total of forty cases in which valvular heart disease and pulmonary tuberculosis existed together in the same individual. Traube and others state that they have often met with aortic disease in cases of advanced tuberculosis, but never or only exceptionally with mitral disease. Krygen's ten cases of valvular heart lesions met in 1100 sections in cases of tuberculosis furnished four instances of aortic incompetency, three of mitral incompetency, and two of pulmonary stenosis. For this discrepancy of opinion and experience of different observers there is no satisfactory explanation at present writing.

4. *The effect of right-sided valvular heart disease upon the development and progress of pulmonary tuberculosis.* A careful review of the literature of this aspect of my theme seems to indicate that stenosis of the pulmonary artery predisposes to pulmonary tuberculosis. Stölker[24] noted sixteen instances of congenital stenosis of the pulmonary ostium. He calculates that in the cases of pulmonary stenosis that live to the age of twenty, 14 per cent. succumb to pulmonary tuberculosis. Lebert's[25] estimate, however, is much higher, or 33 per cent. of all cases surviving the twentieth year. This observer[26] also gives the literature of twenty-four cases of stenosis of the conus arteriosus of the pulmonary ostium and of the pulmonary artery, all of which (fifteen males and nine females) were complicated with phthisis. Bernard Schultz[27] found six cases of stenosis, five of which revealed complicating pulmonary tuberculosis. Constantin Paul[28] has examined, post-mortem, thirty-three cases of congenital stenosis of the pulmonary artery, and has formed the conclusion that phthisis commonly develops as the result. Heftler[29] believes that congenital heart affections, particularly pulmonary stenosis, favor the coincidence of morbus cordis and pulmonary tuberculosis.

While congenital stenosis of the pulmonary artery is often followed by tuberculosis, it must be recollected that, independently of the latter disease, life is prolonged beyond the second decade only under the most

careful arrangement of all the sanitary details. The dangers that beset such subjects are numerous and often disastrous in their results. The conditions are not favorable to healthy nutritive processes, and the lungs must share disproportionately, owing to the insufficient blood-supply, in the general nutritive disturbance, and thus become unduly susceptible to tubercular invasion. Obviously, the lungs must remain more or less undersized and ill-nourished from the date of birth—a condition that explains in a satisfactory manner the increased tendency to tuberculous infection. The extreme dyspnœa and cough from which these unfortunates at last suffer upon attempting muscular exercise compels them to keep quiet, and, in consequence of this interference with ordinary movements, the strength declines and the general health becomes greatly impaired. Neither is it judicious in the treatment of these cases to advise any but the slowest and most cautious muscular exercise. The foregoing facts explain the rather remarkable tendency of the subjects of pulmonary stenosis to contract phthisis, but its influence as an indirect cause of the latter disease, in view of the available testimony at hand, has probably been somewhat overrated.

We are not concerned here especially with the question of the diagnosis of cases of pulmonary stenosis, but I desire to draw attention to the fact that a myocarditis of the conus of the ventricle, as first pointed out by Dittrich, may also induce narrowing of the pulmonary orifice; hence the diagnosis of valvular endocarditis cannot always be made with a feeling of absolute assurance.

Lebert[30] has collected five cases from the literature in which the pulmonary stenosis was located in the right conus arteriosus, and these cases manifest a similar tendency to consecutive pulmonary tuberculosis. On the other hand, it is interesting to note that in cases in which communication exists between the ventricles, without associated pulmonary stenosis, phthisis is rarely encountered.

The clinical differences between phthisis in the advanced stage and combined pulmonary stenosis and phthisis are not so obvious as at first sight appears. The dyspnœa, cyanosis, the right ventricle hypertrophy may be duplicated in pure phthisis. That considerable hypertrophy of the right chamber is generally present in pulmonary tuberculosis may be easily verified by fluoroscopical examination. Not to be overlooked in the discrimination of the hybrid from pure phthisis are the bellows murmur or sound, which is to be heard at the pulmonary artery valve, and is transmitted upward and to the left, and the previous history, which clearly points to the presence of the dyspnœa and cyanosis from the time of suckling.

I append here the literature of thirty-six cases (exclusive of the twenty-four given elsewhere by Lebert[31]) in which phthisis developed secondary to pulmonary stenosis.

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REPORT OF A CASE OF DEMENTIA PRÆCOX.

By William Rush Dunton, Jr., M.D.,
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The following history is imperfect in many ways, but seemed to the writer to have sufficient points of interest to warrant publication. Dementia præcox is not well known in this country, and the present case shows very well the physical characteristics of the disease. The mental characteristics have not been noted as fully, partly because the writer was not sure of certain phases apparently shown by the patient and partly to avoid making this report too long.

Mrs. G. T. was admitted to the Sheppard and Enoch Pratt Hospital July 1, 1899. She was then thirty-one years of age, born in Norway and married, having three children, the youngest being eighteen months old. Family history is negative. There was no history of any serious illness excepting that after the birth of her last child she had chronic bronchitis, which lasted nearly all winter, but from which she recovered entirely.

The first mental change was noticed in the latter part of May, 1899, when she went to communion, and afterward she said that she had done wrong in attending the service. This seemed to worry her very much, and she accused herself as well as her husband of wrong-doing. She then began to neglect her household duties, to sit about and brood. She thought that people would kill her, burn her up, or cut her in pieces. She complained of the street noises and said she was frightened. She wanted her husband to stop the noises and said she could not endure them. Some days she was brighter than others, but there was little real change in her condition. A few days before admission she said that if she were left alone in the house she would burn it up. Her physical symptoms were as follows: Her periods had been regular, she had been sleepless, had lost flesh, although she had been eating fairly well, and had been constipated. On admission there was but little change in her symptoms until about a month after admission, the only change

being that her appetite began to fail. About August 1st she refused to answer questions and would merely shake her head. Early in August she began to cough, later had to be put to bed, to be fed and dressed, was very dull and stupid, would let saliva run out of her mouth, and seemed to have pain in her chest, though physical examination showed nothing. August 31st she was put on iodide of potash, 5 grains, t. i. d., and five days afterward seemed a little brighter, and lifted her head when spoken to. Later the iodide was increased to 10 grains, but was stopped after a time, as no improvement followed. Early in September she began to have an offensive discharge from the nose, apparently simply due to her not cleaning it.

In the middle of October she became rather resistive, began to pinch her mouth up tight (snouting), would not eat for a few days, and paid no attention to her friends when they came to see her. About this time the katatonic symptoms were first noticed. She would hold her arms for some time in any position in which they might be placed, but would gradually change the position until it became more comfortable. The symptoms did not change materially, and early in February of 1900 her condition was as follows: Patient antagonizes every attempted passive movement. The resistance includes the facial muscles, and when one attempts to separate the eyelids with the fingers there is great resistance, and if the lids are parted the eyeballs are found rolled upward. Slight percussion over facial nerves causes short, sharp contraction of orbicularis palpebrarum and well-marked twitching over whole area supplied by facial nerves. All muscles are in a state of slight but well-marked tonic contraction which relaxes on stimulation. Fingers and thumbs are tightly flexed. Flexion is not relaxed by irritation with point of needle on other parts of the body. Triceps reflex present, slightly subnormal, periosteal absent, knee jerks and masseter active. Plantar flexion of all toes relaxed, sometimes almost entirely, by tickling soles of feet, and when this is done she tries to draw her feet away. Facial innervation equal on both sides.

Cold baths were tried without any apparent benefit. Her hands, which were flexed constantly, became sore, and it was quite difficult to wash them and apply bandages, as she screamed with apparent pain when they were extended. When anyone talked to her she puckered and pursed out her lips. On February 14th it was noted that she laughed to herself, and also laughed when the nurses spoke to her.

February 22*d*. She had an attack in which she shook as though having a chill. The pulse was 144. During this attack her muscles were not contracted. In March she attacked a nurse, and then spoke for the first time in several months. Later in the day she again attacked a nurse and was very noisy. After this it was possible to have her up and dressed. She talked quite well at times, still had to be fed, and sat very quietly, rarely changing her positon. In May she was well enough to be taken out walking, but generally had to be pushed forward while she took steps, which were about six inches long. At one time she jumped along like a toad. This condition continued, she having a few more excited attacks, and in the middle of October her condition was as follows: October 18, 1900. Patient sitting in chair, put to bed for examination, dorsal decubitus, with knees drawn up, eyes closed, and hands clenched. When addressed does not open eyes, and usually makes no sign of response, but sometimes laughs quietly. Resists any

attempt to open eyelids. Will not open mouth when asked. Does not object to a fly walking all over her face. When touched lightly with a camel's-hair brush about the eyes there is slight movement of the lids, but no attempt to move the brush with the hand and no change of expression. There is no reaction when the patient is touched about the corners of the mouth and no voluntary muscular movement, except at times a slight laugh. When alone the patient sometimes opens her eyes a little, but closes them again when spoken to or when anyone approaches her. At times cries out and has occasionally spoken the names of members

FIG. 1.

of her family, but is usually silent, not even mumbling. When tickled with a brush in the external ear there is slight contraction of the external ocular muscles and slight action of the zygomatici, invariably accompanied by a slight chuckle. Patient does not attend to bodily needs, soils herself if not watched. Is able to drink from a glass held in both hands without spilling the contents, and eats with a spoon held invariably in the right hand. Has slight muscular memory in distinguishing right from left. Strong pain stimuli do not seem to produce any reaction. Patient does not wince when the skin of the forearm is severely pinched. Auditory stimuli, on the contrary, are quickly

responded to. Head can be turned from side to side without resistance and the arms placed in different positions without opposition. The hands can be opened, but fingers cannot be fully extended. Thumb can be moved freely, but there is contracture of the interossei and lumbricales of both hands. When the terminal phalanges are forcibly extended there is a slight movement of the head which suggests the possibility that the forced movement causes the patient pain. There is a tendency for the arms, when placed in unnatural positions, to remain a considerable length of time as they are placed, and then they gradually drop. There is no tendency to grasp objects thrust into her palms. When tapped lightly on the face with percussion hammer there is marked movement of the eyelids, more so than when touched with the brush. Percussion of the regions supplied by the seventh nerve shows marked mechanical irritability of the muscles. No marked tenderness over skull on percussion. Eyeballs are apparently rotated upward, although it is almost impossible to separate the lids. Patient is not careful to be cleanly, and, when suffering from coryza, often allows mucus to appear and run down over her face.

Superficial circulation very poor. Passive movements of legs and feet are not resisted. Lies with toes turned in. Inner border of soles slightly elevated. When tickled on the sole of the foot there is no response. Knee-jerks exaggerated. When sole is scratched with a pin there is slight movement to withdraw the foot. Pin scratching causes plantar flexion of all the toes. Patient never dresses herself and has no initiative, but when very excited has sometimes taken off nearly all of her clothes. While sitting in bed scapulæ are rotated outward from mid-line, and inner border is raised from chest wall; both deltoids are very poorly developed, leaving prominences of shoulders plainly visible.

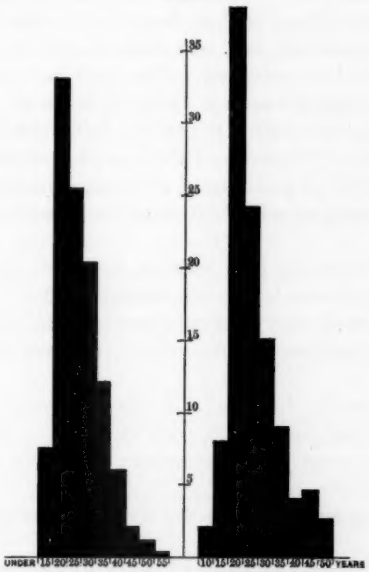
There is some atrophy of the supraspinous region. When uncovered patient begins to shiver as though cold, but when asked to nod her head if cold she replies by shaking her head. Pectoral muscles very poorly developed. Resisted having breast exposed.

With the galvanic current there is a short, sharp contraction of the right deltoid, left being somewhat sluggish. On stimulating the facial nerve there is a short, quick contraction with a current of three milliampères. No increased mechanical irritability of muscles, and no marked response on Erb's point. With a faradic current of sufficient strength to give fairly strong contraction of muscles there is no evidence of sensation.

September 21, 1901. The patient was to-day discharged unimproved and taken to one of the State hospitals. Her condition had not changed since last note. She was in bed a great deal because she seemed more comfortable, was less apt to have sudden attacks of violence, and was more easily cared for. Menses were regular throughout her stay in the hospital. She weighed ninety pounds when discharged, a loss of thirty-eight and one-half pounds since admission. In the past year she had lost but three and one-half pounds. After being made ready for discharge the patient would not walk nor stand up, but after a few minutes walked when supported and pushed foward by two nurses, and later stood without support. She answered questions with nods and shakes of her head, but would not speak. Her hands were blue and fingers were immovably semiflexed.

Kraepelin gives the following symptoms as common to all forms: The tendon reflexes are increased; the pupils are dilated and may be different in size; saliva is increased; the activity of the heart is weakened; cyanosis and hyperidrosis are often present; the menses are skipped or irregular; sleep is disturbed; the capacity of taking nourishment varies, but the weight decreases.

FIG. 2.



Relative frequency of dementia præcox. Relative frequency of the maniacal depressive insanities (mania, melancholia, stupor). (After KRAEPELIN.)

The simple appreciative perception of external conditions is not interfered with, therefore orientation is undisturbed, but may be dimmed in stupor or influenced with insane ideas when excited.

Sensory perceptions are, however, frequently interfered with, and the order in which they are generally affected is: first, hearing; second, seeing; third, feeling. In the beginning the fallacious sense-perceptions are generally disagreeably, but later may be indifferently or pleasantly interpreted. For example, the patient often thinks he is in a theatre, and may ask questions about the performance. These questions are, of course, foolish or incoherent.

Fixation is poor. After stupor great curiosity may be shown. Negativismus is frequently present. (Negativismus, or negativism, Kraepelin

defines as the senseless struggling against every external influence. It is shown in the mutism or the senseless dumbness, as well as in the complete inability to influence the patient.)

Time-sense is well marked. The power of retaining words, numbers, etc., is generally well preserved, but the power of putting together new words, or of using new words, is diminished, and that the train of thought suffers is shown by stereotypie, rhyming, or verbigeration.

Judgment is always impaired and the sense of right is lost. The will power is lost. There is disturbance of the emotional life. The patient is sometimes sad, and sometimes lively or excited, but the excitement is simply a motor one. They may have sudden attacks of boisterousness. Ideas of beauty are lost. There is no technical ability, and the power to work suffers, the patient being able to do old things, but nothing new. Epileptiform attacks occur, and are more common in men, occurring in 18 per cent. of all cases. Besides this there may be hysterical attacks, apoplectiform attacks, or paralyses, or chorea-like movements.

Tromner says that the four most characteristic abnormalities are: 1, a peculiar confusion in speech, writing, and demeanor; 2, the tendency to the production of bizarre anomalies in gait, gesture, mien, and action; 3, weakness of judgment; 4, termination in the peculiar dementia.

Christian makes the following summary of symptoms: 1, a constant appearance at puberty; 2, various delirious symptoms at the beginning; 3, constant sudden impulses; 4, a rapid termination in a dementia more or less complete.

The majority of authors agree that dementia præcox is a degenerative psychosis.

A summary of the case shows that the patient was mentally depressed. There was an exaggeration of the tendon reflexes, a weakening of the heart's action, cyanosis, and a decrease of weight while taking nourishment well. At one time she refused food and had to be fed, later she took nourishment well. Simple perception of external ideas was not interfered with, but there was fallacious sense-perception, as was evidenced by the early complaints of street noises, etc. Negativism, while present, was not especially marked. There was disturbance of her emotional life, as was shown by her periods of depression and attacks of boisterousness. Stereotypie and verbigeration were shown on several occasions. Katatonic rigidity was also present.

In view of these symptoms the case seems to be the katatonic form of dementia præcox. It is to be regretted that her previous history should be so meagre, but there was a good deal of difficulty in obtaining the little which is given. The age, thirty-one years, at which onset is noted is somewhat uncommon, being beyond the period of puberty,

which Christian has placed between the ages of fifteen and twenty-five. Kraepelin, in his *Text-book of Psychiatry*, gives a diagram showing the percentage of cases in reference to the age of occurrence. More than 60 per cent. occur before the twenty-fifth year, but over 10 per cent. occur before thirty-five years. Tromner, of Hamburg, in his monograph on dementia præcox, has placed this diagram beside one which shows the occurrence of the maniacal-depressive forms (mania, melancholia, stupor) in the same periods of life. It shows very strikingly that dementia præcox is not so essentially a puberty psychosis as was supposed, and that the maniacal-depressive forms are more common in early life than many of us have thought.

The condition of tonic muscular contraction shown by this patient has not been much studied. It occurs without accompanying mental symptoms, but the correlation between the two is practically unknown. It is hoped that physicians may become interested in this condition, and by reporting cases, either with or without accompanying mental symptoms, add to our knowledge of the subject.

The literature on dementia præcox is large, but unfortunately most of it is by foreigners, so that it is not easily reached by all physicians. A good deal has been translated, however, and appended is a list of a small part of the literature.

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ON SARCOMA OF THE RADIX LINGUÆ,[1] WITH REPORT OF A CASE.

By George E. Shambaugh, M.D.,
of Chicago.

In September, 1900, the following case came for treatment to the dispensary at the Woman's Medical College:

A. S., a laborer, aged thirty-eight years, was born and raised in Poland. Father and mother died at the ages of fifty-seven and fifty-

[1] When the mouth is opened as wide as possible and the tongue protruded one may see by direct inspection, in a favorable case, several of the circumvallate papillæ lying near the border of the tongue. These papillæ are arranged in the form of a V, with the apex pointing toward the back and terminating at the foramen cæcum. The part of the tongue in front of

five, respectively; causes not known. Patient had been married sixteen years, and had had five children. One child died in infancy, and the other four are living and well. Previous to his present trouble he had always been well and strong. He was a strongly-built man, of medium height. When first seen, on September 8, 1900, he showed a well-marked cachexia. His face appeared somewhat shrunken, his muscles were flabby, and his skin sallow. His facial expression was strained and anxious, his chin was slightly elevated, allowing the throat in front to protrude, frog-like, as when the base of the tongue is strongly depressed.

His throat trouble began eight months previous, with slight pain on swallowing, in the region of the hyoid bone. With the exception of this trouble on swallowing, he had never suffered any pain, but had been troubled a great deal with mucus accumulating in his throat.

FIG. 1.

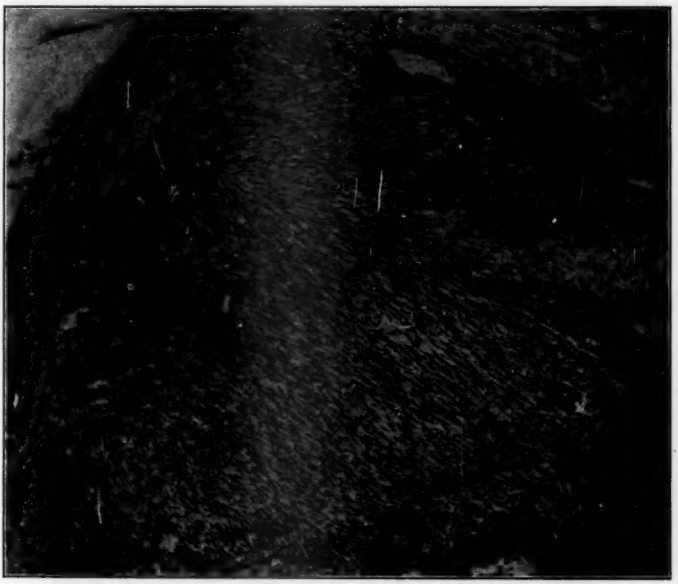
This he had great trouble in dislodging. He first experienced difficulty in swallowing food, two months previous. After a time he could swallow solid food only after he had pushed it past the swelling with his finger. Later he was unable to swallow solid food at all, and for the past two weeks he had had trouble in swallowing liquid food on account of the regurgitation which took place through the nose. His respira-

the circumvallate papillæ is developed (according to His) from the tuberculum impar, which springs from the median line of the floor of the pharynx between the first and second arches. The part of the tongue back of these papillæ results from the growing together of two lateral halves, which spring from the antero-lateral pharyngeal walls in the location of the second and third visceral arches. This posterior part of the tongue, having an origin quite distinct from the anterior part, is called the radix linguæ (Anatomische Nomenclature, Basil, 1895). The term "base of the tongue" has sometimes been applied to it also.

tion had been interfered with somewhat, and his voice was greatly altered by the mechanical obstruction in the oropharynx.

Examination of the neck disclosed no glandular enlargement. Inspection of the oral cavity showed the front part of the tongue normal. The posterior part of the oral cavity and the oropharynx were occupied by a tumor the size of a small hen's egg, lodged on the left side, hiding the pillars of the fauces and the soft palate on that side. (Fig. 1.) At times the soft palate was seen stretched over the rounded top of the tumor, which then projected into the postnasal space. This tumor had an oval outline, its surface was smooth and of a red flesh color, with dilated veins showing at several places. It felt soft, and bled readily when disturbed

FIG. 2.

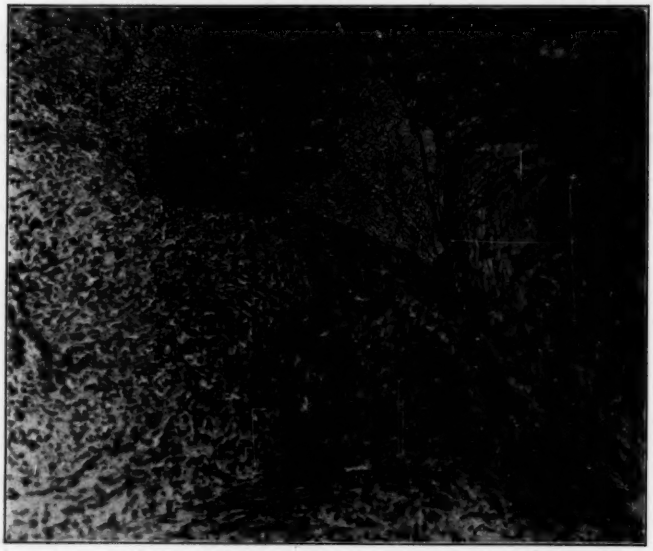


Showing spindle-cells and bloodvessels. × 60.

with a probe. Its attachment appeared, on palpation, to be deep down on the radix linguæ. Occupying the middle and right thirds of the radix linguæ was a dense, firm, elastic mass, elevated about one-quarter of an inch above the surface of the tongue, extending as far forward as the circumvallate papillæ, and with its anterior edge somewhat curled over its attachment. Its nodular surface was covered with smooth mucous membrane. It did not bleed so readily as the tumor on the left. The presence of ulceration was not detected on any part of the growth.

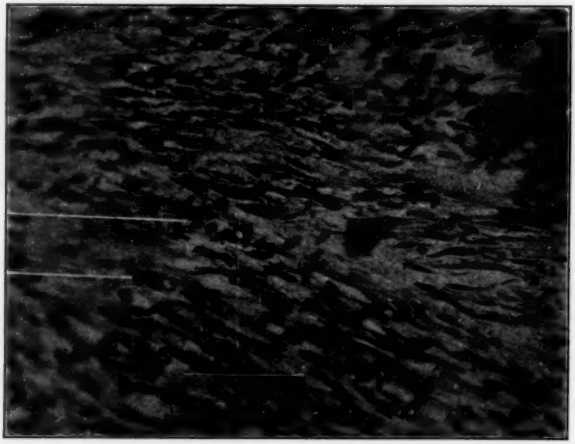
The patient was seen again on September 30th, when he said that a week previous he had suffered a severe hemorrhage from his throat,

FIG. 3.



Showing spindle-cells cut in cross section, bloodvessels, and extravasation of the blood into the tissue. × 90.

FIG. 4.



a b

Showing large spindle-cells separated by intercellular substance; also karyokinetic figures (*a*, *a*, and *b*, *b*). × 200.

when he had lost two quarts of blood. He had been very weak and dizzy and had tinnitus aurium following the hemorrhage. He appeared much paler than when seen before. The large tumor on the left was slightly shrivelled. It had partially lost its red flesh-color and was somewhat yellowish. At this time I removed from the mass on the right a small piece for microscopical examination.

This piece was made up of large spindle-cells, arranged in broad currents streaming in different directions through the tumor. (See Fig. 5.) No distinct epithelial covering could be made out, nor was there a distinct capsule. At one place only was there a structure that resembled a capsule. (See Fig. 2.) In several places large areas of necrosis were found. Scattered through the middle of the section were a number of large, open spaces filled with blood, evidently extravasa-

FIG. 5.

Showing streams of spindle-cells, numerous bloodvessels, and extravasation of blood. × 60.

tion of blood into the tissue. (See Figs. 3 and 5.) The section was quite rich in bloodvessels. The cells were large, fusiform, or spindle-like, many having elongated thread-like ends. (See Fig. 4.) In many of the cells, karyokinetic changes could be seen. (See Fig. 4.) The cells, for the most part, were not packed closely together, but were separated by a considerable amount of fibrous intercellular substance.

The tumors which involve the radix linguæ are rare, if we exclude the not uncommon hypertrophy of the lingual tonsil. The benign tumors that have been found in this locality include fibroma, fibro-lipoma, fibromyoma, chondroma, cysts, adenoma, accessory thyroids, dermoids, gumma, and cavernoma. Of the malignant tumors, primary carcinoma appears to be the most rare, while secondary carcinoma is

relatively not uncommon. Sarcoma of this region, while not so rare as primary carcinoma, is of exceedingly rare occurrence. I have been able to collect but nine cases from the literature on the subject. The first case was reported by Fiedler as early as 1864. No other case was reported until 1885, twenty-one years later, when Albert reported his case of round-celled sarcoma of the basis linguæ. The following year, 1886, Eve examined microscopically and described an old museum specimen of the Hunterian collection, which proved to be a round-celled sarcoma. The next year Beregszaszy reported a case of lymphosarcoma. In 1888 Schulten reported a case of small-celled sarcoma. In 1892 Scheier reported one of small round-cells, and in 1894 Perman reported one of round-cells. In 1895 Onodi published the case of a round-celled fibrosarcoma occurring in a girl, aged seventeen years. Michael, in 1895, reported the only case of secondary sarcoma of the radix linguæ that has been published.

In this list I have not included the following cases, where doubt exists as to the involvement of the radix linguæ.

A case reported by Gross,[1] in 1872, of spindle-celled sarcoma, occurring probably congenitally in a child, where the tumor is described as occupying the posterior half of the dorsum of the tongue, but it is doubtful whether the radix linguæ itself was involved.

A case reported by Butlin,[2] in 1887, where the tumor occupied the attachment of the tongue (root of the tongue—Butlin), causing a bulging both above and below this organ. He describes it as extending from the junction of the anterior and middle thirds to the back of the tongue, but is not clear as to the involvement of the radix linguæ.

Regarding the etiology of sarcoma of the radix linguæ, nothing definite can be formulated from the cases reported. In one case the irritation of a broken tooth and of excessive cigarette-smoking was given as a possible cause. In Scheier's case it looked as if the sarcoma might have developed in the base of a previously existing ulcer. Cases of sarcoma in other parts of the tongue, developing in the seat of an ulcer, have been reported by Dunham[3] and by Littlewood.[4] Heredity does not seem to play any part in its occurrence. In one case only was the sarcoma secondary. In this case the primary growth appeared in the tonsil. It occurs with equal frequency in either sex. Of the nine cases where the sex has been given, four were female and five were male. It appears to occur at any age. Of the nine cases where the age has been given, one occurred at the age of seventeen, and one at the age of fifty-six. All the other cases occurred between the ages of

[1] Philadelphia Medical Times, 1887, vol. ii. p. 272.
[2] Lancet, March 26, 1887, p. 632.
[3] The American Journal of the Medical Sciences, Philadelphia, 1895, n. s., vol. cx. p. 259.
[4] Transactions Pathological Society, London, 1897-98, vol. xlix. p. 60.

twenty-eight and forty-two. In the case reported by Gross[1] the tumor appeared to be congenital. Marion,[2] who collected twenty-one cases of sarcoma of the tongue, in which the ages of the patients were given, found that twelve occurred below thirty years, three between thirty and forty, three between forty and sixty, and three over sixty.

Sarcomata of the radix linguæ have been found from the size of a hazelnut to that of a hen's egg. They may be distinctly encapsulated or appear as diffuse infiltration of the tissue from which they take their origin, or they may appear pedicled. The surface is usually smooth, but may appear nodular, and is usually covered with normal mucous membrane. In two cases only was ulceration detected. Its consistency varies from the firm elastic tumor of the fibrosarcoma to that of semi-fluctuation. The tumor may be of one piece, or it may be distinctly lobulated. In no case was there a definite history of the existence of more than one tumor. In the case reported by me, it looked as if we had to do with two distinct tumors.

Histologically, all of the cases where the type of cells was specified are cases of round-celled sarcoma, except Fiedler's case and the one which I report, which are cases of spindle-celled sarcoma.

The symptoms caused by sarcoma of the radix linguæ are very similar to those caused by any benign tumor in this locality, symptoms referable chiefly to the mechanical disturbance caused by the size of the growth. In the beginning, sarcoma often causes but an unpleasant sensation in the throat, associated with an increase in the amount of mucus secreted. As the tumor develops, pain and difficulty in swallowing solid food are noted. Later the voice is impaired, and respiration interfered with. Ulceration is not common. In only two of the cases was it noted. Glandular involvement is rare, as is the case with sarcoma in other localities. When it has been found, it was referable, as a rule, to local infection rather than to the extension of the sarcomatous growth to the glands. Metastasis to the peritoneum was noted in one case. Pain, which is such a prominent symptom in carcinoma, is not pronounced in sarcoma, except where ulceration exists. Hemorrhage is also rare, owing to the absence of ulceration. The course of the disease is usually quite rapid. In only three of the cases had the tumor existed more than one year after the first symptoms appeared, while in four of the cases it had lasted less than six months.

The diagnosis of sarcoma of the radix linguæ from the benign tumors which are found here is not, as a rule, difficult. The wide insertion of the sarcoma and its rapid growth marks it as in all probability malignant. Its color more nearly resembles that of the surrounding tissue than is the case with benign growths. When the sarcoma is large its

[1] Loc. cit.

[2] Rev. de Chir., Paris, 1897, vol. xvii.

growth is such as to alter the contour of the surrounding parts, while a benign tumor is more apt to conform to its surroundings and to cease growing when it meets an obstruction. From carcinoma the diagnosis is not always easy to make. When we have to deal with the non-ulcerating variety of sarcoma this clinical fact alone is strong evidence that the tumor is not carcinoma, for the latter almost always breaks down quite early. With the ulcerating variety, on the other hand, the question is more difficult. The absence of marked pain and glandular enlargement would distinguish the sarcoma from carcinoma where both of these symptoms are usually marked. It should be noted here that with an ulcerating sarcoma pain is much more apt to be present than in the non-ulcerating variety, and that in this type of sarcoma, glandular enlargement from infection is quite apt to be present. Clinically the diagnosis cannot always be made between the two types of malignant tumors. In Beregszaszy's case a diagnosis of cancer was made until a post-mortem examination showed the case to be sarcoma. In these cases of ulceration the histological diagnosis is not always easy to make, as we may repeatedly succeed in getting only a piece of the granulating tissue about the sarcoma for examination, as happened with Scheier before he finally was fortunate enough to get a piece of the sarcoma.

Syphilis may readily be mistaken for sarcoma, especially in the pre-ulcerative stage. Esmarch[1] expressed his belief that most of the cases diagnosed as sarcoma of the tongue were cases of syphilis, and would clear up under the proper treatment.

After ulceration has occurred the diagnosis may still be difficult, for sarcoma may also ulcerate, and it is right here again where the histological examination is likely to fail one. For these reasons, where any doubt exists as to the nature of the growth the diagnosis should be reserved until the case had been subjected to vigorous anti-syphilitic treatment. It should be remembered that sarcoma, too, may show a temporary improvement under the administration of potassium iodide.

The prognosis is not so unfavorable as one might at first suppose. Of the nine cases where the clinical history is given, four received no treatment. Two of these were lost sight of, and the other two were considered inoperable cases, and died from exhaustion. Of the remaining five cases, in two, where the tumor was removed, recurrence and death took place; in two cases where the tongue was removed one died of pneumonia eight days later, the other got well, and three years later there had been no recurrence. The fifth case was cured by injections of pyoktanin and subsequent removal of the tumor with the galvano-cautery snare. In the case reported by Butlin,[2] where the sarcoma

[1] Langenbeck's Archives, Bd. xxxix., Heft 2.

[2] Loc. cit.

occupied the left side of the posterior half of the tongue, the left half of the tongue was removed. The case recovered, and four years later there had been no recurrence.

Surgeon.	Age	Sex	Duration.	Histology	Pain.	Ulceration.	Involvement of glands.	Treatment.	Result.
Fiedler.[1]	40	M.	Several years.	Soft spindle-celled sarcoma.	Yes.	.....	Yes.	Extirpation with galvano-cautery snare	Recurrence and death from exhaustion.
Albert.[2]	56	F.	3 yrs.	Round-celled sarcoma.	No.	No.	No.	Tongue extirpated.	Died of pneumonia eight days later.
Eve.[3]	adt.	...	.....	Round-celled sarcoma.					
Beregszaszy.[4]	42	M.	2 mos.	Lympho-sarcoma.	Yes.	Yes.	Yes.	No treatment	Developed metastasis in peritoneum eight days later.
Schulten.[5]	32	F.	½ yr.	Small celled sarcoma.	No.	No.	No.	Extirpation of tumor; recurrence; tongue extirpated by suprahyoid pharyngotomy.	Three years later no recurrence.
Scheier.[6]	28	M.	2 yrs.	Small round-celled sarcoma.	Yes.	Yes.	Only after operation.	Tumor removed.	Recurrence and death.
Onodi.[7]	17	F.	7 mos.	Round-celled fibro-sarcoma.	No.	No.	No.	No treatment	Patient lost sight of.
Perman.[8]	30	F.	6 mos.	Sarcoma.	No.	No.	No.	Injections of pyoktanin and subsequent removal with snare.	One year after removal no recurrence.
Michael.[9]	30	M.	Few weeks.	Lympho-sarcoma-	...	.....	Yes.	Inoperable.	Died of cachexia.
The author.	38	M.	8 mos.	Large spindle-celled sarcoma.	No.	No.	No.	No treatment	Patient lost sight of.

[1] Ztschr. f. med. Chir. u. Geburtsch., Leipzig, 1864, n. f. iii. pp. 305-312.
[2] Wien. med. Presse, 1885, p. 171.
[3] Transactions Pathological Society of London, 1886, vol. xxxvii. p. 223.
[4] Krankheiten der Zunge von Butlin. Wien, 1887, p. 226.
[5] Finska. Läkaresällskopets handlingar, 1888, p. 689. Cited in Deutsche Ztschr. f. Chir., Bd. xxxv. p. 417.
[6] Berl. klin. Wochenschr., 1892, p. 584.
[7] Monatschr. f. Ohrenh., 1895, vol. xxix. p. 75.
[8] Hygiea, 1894, vol. i. p. 367; also, Buffalo Medical and Surgical Journal, 1894, p. 148.
[9] Handbuch der Laryngol. u. Rhinol. Heimann. Wien, 1899, vol. ii. p. 634.

A REVIEW OF ECHINOCOCCUS DISEASE IN NORTH AMERICA.

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of Buffalo, N. Y.

The writer's intent in the present article is to present the statistics of hydatid (echinococcus) disease in the United States and Canada, based upon the cases and references collected from the literature and other sources to date—July 1, 1901—and to add thereto certain observations suggested by the facts shown, with the hope that such a study may contribute to the interest and knowledge of this rare disease in America, and make available for reference the cases that are scattered through the mass of medical literature.

Two previous reviews of hydatid disease in America have been published, the first by Osler,[2] in 1882, collecting sixty-one cases from the United States and Canada, and the second by Sommer,[3] in 1895 and 1896, bringing together 100 cases from the United States. Osler[4] states also that Alfred Mann collected for him from the literature in the period—1882, to July, 1891—twenty-four in addition to his previous sixty-one cases. Mann's summary of cases was not published, but Dr. Osler has informed us that all of Mann's cases were later included in Sommer's collection. The statistics of Osler and Sommer together include 110 cases. To these cases we have been able to add 135 other cases, so that our statistics embrace in all 241[5] cases occurring in the United States and Canada.

The statistics, in brief, of the 241 cases are given in Table I., as follows:

Table I.—Summary of Cases of Echinococcus Disease in North America.

Case 1.—Low, 1822. N. Y. Med. and Phys. Journal, i. 287–289. Male, aged twenty-nine years, white, Albany, N. Y. Hydatids in bladder.
Case 2.—Low, 1822. N. Y. Med. and Phys. Journal, i. 287–289. Female, aged seventy-three years, white, Albany, N. Y. Hydatids in liver and ovary.
Case 3.—Alexander, 1838. Boston Medical and Surgical Journal, xviii. 37–42. Male, aged thirty-five years, foreigner, Danville, Vt. Cysts in liver (about 200).
Case 4.—Polk, 1841. West. Journ. Med. and Surg., Louisville, 2 S., iv. 34–35. Male, aged sixty-five years, white, Perryville, Ky. Liver.

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[1] Read at the annual meeting of the New York State Medical Association, held at New York, October 21–24, 1901.

[2] Osler. The American Journal of the Medical Sciences, October, 1882, N. S., vol. lxxxiv. pp. 475–480.

[3] Sommer. New York Medical Journal, November, 1895, vol. lxii. pp. 656–659, and August, 1896, vol. lxiv. pp. 262–265.

[4] Osler. Practice of Medicine, New York, 1895, second edition, p. 1102.

[5] We have omitted from our statistics four of Osler's and Sommer's cases, as they seemed of doubtful validity.

Case 5.—Gross, 1845. Path. Anatomy, Phil., 2 Ed., 662. Male, aged thirty-five years, Cincinnati, O. Large cyst in liver; rupture into peritoneal cavity.
Case 6.—Moses, 1846–47. Annalist, N. Y., i. 253–255. Male, adult, Governor's Island, N. Y. Hydatids in liver.
Case 7.—Granger, 1849–50. Buffalo Med. Journal, v. 80–82. Male, aged sixty years, Lawrenceville, N. Y. Hydatids in ventricles of brain.
Case 8.—Weber, 1852. N. Y. Med. Wochenschr, April. Male, aged twenty-six years, German, New York City. Cyst in liver.
Case 9.—Ellis, 1856. Boston Med. and Surg. Journ., liv. 344, and Extract Rec. Boston Soc. Med. Improv., ii. 332. Female, aged twenty-eight years, Italian, Boston, Mass. Liver.
Case 10.—Ellis, 1856. Extract. Rec. Boston Soc. Med. Imp., ii. 332. Male, aged twenty-four years, Irish, Boston, Mass. Liver.
Case 11.—Evans, 1856. West. Lancet, Cincinnati, xvii. 680–694. Female, aged thirty-four years, Welsh, Newport, Ky. Liver.
Case 12.—Finnel, 1856. N. Y. Journ. of Med., N. S., i. 216, and Trans. N. Y. Path. Soc., iii. Male, aged twenty-seven years, English, New York City. Cyst in liver: no hooklets.
Case 13.—Gross, 1857. Path. Anatomy, Phil., 3d Ed., 609. Fifteen small cysts of liver.
Case 14.—Smith, F. G., 1858. N Amer. Med.-Chir. Rev. (Proc. Path. Soc. Phil.), ii. 333–334. Female, aged thirty-four years, Philadelphia, Pa. Liver; cysts expectorated; discharge of fluid by bowels.
Case 15.—Park, 1858. Nashville Journ. Med. and Surg., xv. 120–125. Female, aged seventy-three years, white, Franklin, Tenn. Hydatids in abdomen.
Case 16.—Gay, 1858. Boston Med. and Surg. Journ, lvii. 218–220. Male, aged seventy-one years, Boston, Mass. Echinococcus in lungs.
Case 17.—Gay, 1858. N. Amer. Med.-Chir. Rev., ii. 506. Female, Philadelphia, Pa. Echinococcus in lung.
Case 18.—Minot, 1859. Boston Med. and Surg. Journ., lxi. 279. Female, aged thirty-five years, Marblehead, Mass. Liver; echinococci expectorated, and passed per rectum.
Case 19.—Sands, 1861. Amer. Med. Times, ii. 376. Female, adult, New York City. Cyst in fascia of neck.
Case 20.—Gross, 1862. System of Surgery, 2 Ed., ii. 968. Hydatid of breast.
Case 21.—Gross, 1862. System of Surgery, 2 Ed., ii. 968. Hydatid of breast.
Case 22.—Simmons, 1864. Pacific Med. and Surg. Journ., San Francisco, ii. 264-266. Male, aged ten years, Sacramento, Cal. Abdominal tumor; echinococci passed per rectum.
Case 23.—Marsh, 1869. Cincin. Lancet and Obstet., N. S., xii. 539–543. Male, aged fifty-three years, Indian Hill, O. Kidney.
Case 24.—Nancrede, 1871. Trans. Path. Soc., Phil., iii. 1866–1876, 146, and Amer. Journ. Med. Sci., N. S., lix. 417–421. Male, adult, Philadelphia, Pa. Hydatid cyst of liver.
Case 25.—Sherard, 1872. Med. and Surg. Rep., Phil., xxvii. 143–145. Female, aged twenty-nine years, Mobile, Ala. Hydatid passed per rectum.
Case 26.—Hutchinson, J. H., 1874. Trans. Phil. Path. Soc., iv. 38–40. Male, aged thirty two years, French, Philadelphia, Pa. Large cyst of liver containing hundreds of daughter cysts; small cyst of liver; large puckered cicatrix in liver; large cyst from connective tissue of pelvis.
Case 27.—Tyson, J., 1874. Trans. Phil. Path. Soc., iv. 98–99. Male, aged thirty-two years, Philadelphia, Pa. Small cyst of liver; scolices. (Phil. Hosp.)
Case 28.—McKinnon, 1874. Amer. Med. Weekly, i. 79–81. Female, Selma, Ala. Bladder.
Case 29.—Ellis, 1874. Boston Medical and Surgical Journal, xc. 553–559. Male, aged forty-three years, Azorian, Boston, Mass.
Case 30.—Osgood, H. R., 1876. Transactions of the Philadelphia Pathological Society. Female, aged eighty-one years, Philadelphia, Pa. Small degenerated cyst of liver; hooklets, cholesterin.
Case 31.—Pierce, Bartholow, Campbell, 1877. Clinic, Cincinnati, xii. 157, 169, 181. Male, aged forty-five years. Indianapolis, Ind. Liver.
Case 32.—Dean, 1877. St. Louis Medical and Surgical Journal, xiv. 420–421, and Transactions of the New York Pathological Society, iii. Female, negro, St. Louis, Mo. Echinococcus multilocularis of liver.
Case 33.—Dean, 1877. St. Louis Medical and Surgical Journal, xiv. 420–424. Male, aged thirty-nine years, Bavarian, St. Louis, Mo. Echinococcus multilocularis of liver.
Case 34.—Fauntleroy, 1878. Virginia Medical Monthly, Richmond, iv. 282–284. Aged forty-five years, Staunton, Va. Hydatids in kidney.
Case 35.—Drude, 1878. Virginia Medical Monthly, Richmond, iv. 666–670. Male, aged forty-four years, German, Quincy, Ill. Liver.
Case 36.—Delafield, 1878. Philadelphia Medical Times, viii. 609–611. Male, aged forty-six years, German, New York City. Hydatids of omentum.
Case 37.—Loomis, 1878. Medical Record, New York, xiv. 281–283. Male, aged forty-three years, Irish, New York City. Hydatids of lung.
Case 38.—Jacobi, A., 1879. Transactions of the New York Pathological Society, iii. New York City. Cyst in liver, degenerated; hooklets and cholesterin. (Bellevue Hospital).
Case 39.—Metcalfe, J. T., 1879. Transactions of the New York Pathological Society, iii. New York City. Degenerated cyst in liver; hooklets and cholesterin. (Bellevue Hospital).
Case 40.—McCready, 1879. Transactions of the New York Pathological Society, iii. Male, old. New York City. Echinococcus in common bile duct; three daughter cysts.
Case 41.—Loomis, A. L., 1879. Transactions of the New York Pathological Society, iii. Male, aged thirty-eight years. New York City. Large cyst in liver; daughter cysts and hooklets.
Case 42.—Keyes, E. L., 1879. Transactions of the New York Pathological Society, iii. Male, English, New York City. Cyst in liver; cyst-shreds vomited and passed per rectum.
Case 43.—Jacobi, A., 1879. Transactions of the New York Pathological Society, iii. Female, aged twenty-nine years. New York City. Large cyst in liver; daughter cysts and scolices.
Case 44.—Cory, D. M., 1879. Transactions of the New York Pathological Society, iii. Male, German, New York City. Cysts in liver; scolices.
Case 45.—Van Buren, 1879. Transactions of the New York Pathological Society, iii. New York City. Cyst in liver.

Case 46.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. New York City. Cyst in liver; daughter cysts.
Case 47.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. Female, adult, New York City. Echinococci vomited from liver or omentum.
Case 48.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. Male, old, New York City. Tumor in common bile-duct; obsolete cysts in liver; no booklets.
Case 49.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. New York City. Liver.
Case 50.—McCready, 1879. Transactions of the New York Pathological Society, iii. New York City. Large cyst in liver; booklets.
Case 51.[1]—Ainsworth, 1880. Medical Record, New York, xviii. 346-347. Male, aged forty years, Polish. Fort Vancouver, Wash. Cyst in lung, spleen, and bladder.
Case 52.—Polk, 1880. Medical and Surgical Reporter, Philadelphia, xliii. 290-291. Female, adult, New York City. Cysts in gastro-hepatic omentum.
Case 53.—Hart, 1880. Cincinnati Lancet and Clinic, N. S. iv. 495. Male, German, Cincinnati, O. Cyst in liver.
Case 54.[2]—Osler, W., 1880. Montreal General Hospital Reports, i. 314. Male, Montreal, Quebec. Cyst in liver; scolices.
Case 55.—Eldridge, 1881. American Journal of Obstetrics, New York, xiv. Female, aged twenty years, Japanese, Washington, D C. Bladder.
Case 56.—Fenger, 1881. American Journal of the Medical Sciences, Oct., 377 Male, aged thirty-seven years, Italian, Chicago, Ill. Cyst in lung.
Case 57.—Pollak, 1881 St. Louis Medical and Surgical Journal, xli. 492-493. Female, aged twenty-six years, St. Louis, Mo. Bladder.
Case 58.—Smith, D. F., 1882. Canada Medical and Surgical Journal, xi. 195-197. Female, aged twenty years, Walkerton, Ont. Lung; discharging through sinus in chest wall behind.
Case 59.—Bernays, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Male, adult, English (from Honolulu), St. Louis, Mo. Echinococci expectorated from lungs.
Case 60.—Bernays, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Female, adult, German, St. Louis, Mo Cyst in liver, rupturing into bowel.
Case 61.—Hyndman, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Cincinnati, O. Echinococcus of liver.
Case 62.—Hyndman, 1882 American Journal of the Medical Sciences, lxxxiv. 475-480. Cincinnati, O. Echinococcus of brain.
Case 63.—Ogden, 1882 American Journal of the Medical Sciences, lxxxiv. 475-480. Female, foreigner. New Orleans, La. Cyst in liver.
Case 64.—Buchan, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Female, adult, Icelander, Toronto, Ont. Cyst in liver; scolices.
Case 65.—Cameron, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Female, Toronto, Ont. Cyst in walls of pelvis; two cysts in liver, rupturing into intestine.
Case 66.—Cameron, 1882. American Journal of the Medical Sciences, lxxxiv. 475-480. Female, young adult, English, Toronto, Ont. Cyst in liver.
Case 67[3].—Black, 1882. Canada Medical and Surgical Journal, xi. 140-144. Male, aged twenty-nine years, English (6 years in Canada), Uxbridge, Ont. Cyst in spleen; suppurating cyst in liver, rupturing into lung; cysts expectorated.
Case 68.—Anglin, W. G., 1882. Canada Journal of the Medical Sciences, Toronto, vii 260-262. Male, aged sixty-two years, English, Kingston, Ont. Obsolete cyst of liver; booklets and cholesterin.
Case 69.—Helm, 1883-1884. New England Medical Monthly, Sandy Hook, Ct., iii. 106-111. Female, aged twenty-six years, English, Sing Sing, New York. Hydatids of liver; rupture into intestine.
Case 70.—Helm, 1883-1884. New England Medical Monthly, Sandy Hook, Ct., iii. 106-111. Female, aged forty years, German, Sing Sing, N. Y. Hydatids of liver; rupture into intestine.
Case 71.—Scheaffer, 1884. Transactions of the Medical Society of Penna., xvi. 474. Male, aged fifty-four years, Lewistown, Pa. Hydatids of liver.
Case 72.—Carson, 1884. St. Louis Courier of Medicine, xii. 455. Male, adult, St. Louis, Mo. Hydatids of liver.
Case 73.—Mayer, 1885. Medical Record, N. Y., xxvii 261. Female, aged thirty-eight years, Hartford, Conn. Liver.
Case 74.—Osler, W., 1885. Transactions of the Pathological Society, Phila., xii. 217-218. Male, aged fifty-eight years, English, Ontario, Can. Cysts passed in urine; booklets.
Case 75.—Weir, R. F., 1885. N. Y. Medical Journal, xli. 311-312. Male, aged twenty-six years, English, New Jersey. Bilobed cyst of liver, containing hundreds of daughter cysts; cyst of mesentery.
Case 76.—Kerr, 1886 Canada Medical and Surgical Journal, xv. 120 Icelander, Winnipeg, Man. Cyst of liver. (Winnipeg Gen. Hosp.)
Case 77.—Whittaker, 1886. Cincinnati Lancet and Clinic, N. S., xvi. 704-716. Male, aged sixty-nine years, German, Cincinnati, O Echinococcus in liver.
Case 78.—Bauer, 1886. Philadelphia Medical Times, xvii. 180. Male, aged thirty-five years, German, Philadelphia, Pa. Liver.
Case 79.—Van Giesen, I. J., 1887. Proceedings of the New York Pathological Society, 160-161. Female, adult, German, New York City. Multiple cysts: liver, omentum, abdominal wall, peritoneum, fascia of psoas muscle (4 cysts) mesentery, and uterus; daughter cysts, lamellated membrane, cholesterin; no scolices or booklets found.
Case 80.—Stavely, A. L., 1889. Montreal Medical Journal, xviii. 148-150. Male, aged forty-three years, Russian (Pole), Reading, Pa Enormous cyst of right kidney; booklets.
Case 81.—Ferguson, F., 1890. Proceedings of the New York Pathological Society, 78. New York City. Cyst of liver; daughter cysts and booklets.

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[1] Specimens (Nos. 1342-44) in the Army Medical Museum, Washington, D. C.

[2] Specimens in the museum of McGill Medical School, Montreal.

[3] Age stated by Osler (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1882, lxxxiv. 476).

Case 82.—Chown, H. H., 1890. Medical Record, New York, xxxviii. 469, and personal communication. Male, aged thirty years, Icelander, Winnipeg, Man. Sixteen cysts of different parts of abdominal cavity (first operation); two cysts in peritoneal cavity and a group of cysts in and below liver (second operation); suppurating cyst of spleen with daughter cysts (third operation).

Case 83.—Freeman, R. G., 1890. Proceedings of the New York Pathological Society, 7. Female, aged forty-four years, Sicilian (3 years in U. S.), New York City. Two small cysts of liver; scolices, hooklets, lamellated membrane, daughter, granddaughter, and great-granddaughter cysts. (Roosevelt Hospital, January, 1889.)

Case 84.—Kinyoun, 1890–1891. U. S. Marine Hospital Report, xlix. 147–149. Male, aged thirty-eight years, Swede, New York City. Cysts in liver, kidneys, and bladder.

Case 85.—Allaben, 1891. North American Practitioner, Chicago, iii. 612–617. Female, aged fifty-two years, German, Argyle, Ill. Multiple cysts in bladder, pelvis, liver, spleen, kidney, omentum, peritoneum, diaphragm, and pericardium; had passed cysts per rectum; hydatids removed from abdominal cavity in 1879.

Case 86.—Shattuck, F. C., 1891. Boston Medical and Surgical Journal, cxxiv. 3–5 and 11. Female, aged twenty-three years, Icelander (American parents), Sandwich, Mass. Large suppurating cyst of liver; daughter cysts.

Case 87.—Knapp, P. C., 1891. Boston Medical and Surgical Journal, cxxiv. 10. Female, aged twenty to thirty years, English, Boston, Mass. Two small cheesy cysts of liver. (Boston City Hospital.)

Case 88.—Shattuck and Bradford, 1891. Boston Medical and Surgical Journal, cxxiv. 5. Boston, Mass. Large suppurating hydatid cyst of liver; daughter cysts. (Boston City Hosp.)

Case 89.—Musser, J. H., 1891. Transactions of the Philadelphia Pathological Society, xiv. 83. Female, old, Philadelphia, Pa. Small cyst of liver; hooklets, cholesterin.

Case 90.—de Schweinitz, G. E., 1891. Transactions of the Philadelphia Pathological Society, xiv. 262. Male, aged twenty years, English, Philadelphia, Pa. Cyst in abdominal muscles of right iliac region; brood-sacs, scolices.

Case 91.—Hatch, J. L., 1891. Transactions of the Philadelphia Pathological Society, xv. 339. Old, Philadelphia, Pa. Cyst in liver and spleen. (Philadelphia Hospital.)

Case 92.—Biggs, G. P., 1891. Proceedings of the New York Pathological Society, 47. Male, aged sixty-two years, New York City. Two small cysts of liver; scolices, hooklets, cholesterin, and lamellated membrane.

Case 93.—Lafleur, 1892. Montreal Medical Journal, xx. 694–695. Male, foreigner, Montreal, Quebec. Cyst of liver; hooklets.

Case 94.—Richardson, 1892. American Journal of the Medical Sciences, Oct., 485–486. Cyst in liver.

Case 95.—Mudd, 1892. American Journal of the Medical Sciences, Oct., 412–422. Female, aged twelve years, St. Louis, Mo. Echinococcus of brain. (Surgically a remarkable case.)

Case 96.—Solis-Cohen, 1892. Sajous' Annual, i., c., 33, and New York Medical Journal (1895), lxii. 763. Male, adult, German, Philadelphia, Pa. Liver.

Case 97.—Solis-Cohen, 1892. Sajous' Annual, i., c., 33, and New York Medical Journal (1895), lxii. 763. Male, adult, German, Philadelphia, Pa. Liver.

Case 98.—Solis-Cohen, 1892. Sajous' Annual, i., c., 33, and New York Medical Journal (1895), lxii. 763. Male, adult, Italian, Philadelphia, Pa. Liver.

Case 99.—Haggard, G. D., 1893. Northwestern Lancet, St. Paul, xiii. 3:5. Male, aged twenty-six years, Icelander, Minneapolis, Minn. Cysts in liver, spleen, mesentery, and pelvis. (Minneapolis City Hospital.)

Case 100.—Stamm, 1893. Medical News, i. 365–369. Female, aged forty-nine years, German, Cleveland, O. Abdominal cavity.

Case 101.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Female, adult, Icelander, Winnipeg, Man. Cyst in abdomen, connecting with liver.

Case 102.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Male, aged twenty-five years, Icelander, Winnipeg, Man. Cyst in liver, rupturing into hepatic duct.

Case 103.—Ferguson, A. H., 1893. Northwestern Lancet, xiii, 41–48. Female, aged ten years, Icelander. Winnipeg, Man. Five cysts in liver.

Case 104.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Male, aged twenty-four years, Icelander, Winnipeg, Man. Two large cysts in liver, containing "five common washbasinfuls of cysts and fluid."

Case 105.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Male, aged thirty-nine years, Icelander, Winnipeg, Man. Three large calcified cysts in liver.

Case 106.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Male, aged thirty-nine years, Icelander, Winnipeg, Man. Two cysts in liver: vomited cysts.

Case 107.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelander, Winnipeg, Man. Cyst in lung.

Case 108.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelander, Winnipeg, Man. Kidney.

Case 109.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelander, Winnipeg, Man, Kidney.

Case 110.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelander, Winnipeg, Man. Peritoneum.

Case 111.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelander, Winnipeg, Man. Peritoneum.

Case 112.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Male, Icelander, Winnipeg, Man. Testicle.

Cases 113–127.—Ferguson, A. H., 1893. Northwestern Lancet, xiii. 41–48. Icelanders, Winnipeg, Man. Liver. (Fifteen cases in addition to those above given; no details.)

Case 128.—Biggs, G. P., 1893. Proceedings of the New York Pathological Society, 95. Male, aged thirty-five years, Italian, New York City. Large cyst of liver; daughter cysts, scolices and hooklets.

Case 129.—Herff, 1894. Texas Medical Journal, Austin, ix. 613–616, and xv. 237–245 (1899). Child, San Antonio, Texas. A great many echinococci in bladder; hooklets.

Case 130.—Herff, 1894. Texas Medical Journal, Austin, ix. 613–616, and xv. 237–245 (1899). Female, adult, San Antonio, Texas. Passed per rectum, probably from liver; hooklets.

Case 131.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Bulbus of eye; hooklets.
Case 132.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Extensor muscles of thigh; hooklets.
Case 133.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Liver; one quart of fluid; hooklets.
Case 134.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Liver; two quarts of fluid; hooklets.
Case 135.—Hartwig, M., 1894. Medical Record, N. Y., xlvi. 167. Male, Italian, Buffalo, N. Y. Cyst of abdomen, probably connecting with liver.
Case 136.—Page, 1895. Medical Record, N. Y., xlviii. (September). Female, aged fifty-one years, German, Hiram, O. Liver, pleura, omentum, mesentery, peritoneum, and bladder.
Case 137.[1]—Van Cott, 1895. New York Medical Journal, lxii. 656-659. Mexican, Brooklyn.
Case 138.—Frank, J., 1896. American Journal of the Medical Sciences, cxii. N. S., 437-443. Female, aged thirty-two years, Russian, Chicago, Ill. Large cyst of liver.
Case 139.—Frank, J., 1896. American Journal of the Medical Sciences, cxii. N. S., 437-443. Male, aged twenty-eight years, German, Chicago, Ill. Large cyst of liver, containing hundreds of daughter cysts.
Case 140.—Hughes, W. E., 1896. Transactions of the Philadelphia Pathological Society, xvii. 53. Male, aged thirty years, negro, Philadelphia, Pa. Small cyst of liver.
Case 141.—Custis, 1896. New York Medical Journal, lxiv. 363-365. Adult, Washington, D. C. Vomited (or expectorated?).
Case 142.— ———, 1896. Philadelphia Evening Star, June 13. Male, Muskegon, Mich. Liver.
Case 143.[2]—Keyes and Busch, 1896. Buffalo Medical Journal, xxxv. 25-29. Male, aged fifty-three years, German, Buffalo, N. Y. Multiple cysts in lung, omentum, mesentery, subdiaphragmatic, left kidney, and sac of left inguinal hernia; scolices and hooklets.
Case 144.—Cordier, 1896. Langsdale's Lancet, Kansas City, i. 130-132. Female, aged forty-six years, Kansas City, Mo. Liver; sterile.
Case 145.—Lloyd, 1896. American Med.-Surg. Bull., N. Y., x. 659-661. Male, aged fifty-seven years, Dane, New York City. Muscles of back; about 250 cysts.
Case 146.—Cheney, W. F., 1897. Archives of Pediatrics, N. Y., xiv. 851-855. Male, aged seven years, Italian, San Francisco, Cal. Two large cysts in liver; daughter cysts.
Case 147.—McNamara, S. J., 1897. Medical News, New York, lxxi. 694-695. Female, aged thirty-one years, Italian (9 years in U. S.). Brooklyn, N. Y. Large cyst in upper part of abdominal cavity, probably liver, containing 26 pounds of contents and thousands of daughter cysts; omentum studded with "secondary deposits."
Case 148.—Larkin, J. H., 1897. Proceedings of the New York Pathological Society, 62-63. Male, aged eighty-three years, New York City. Large calcareous cyst of liver, with diaphragm adherent and forming upper part of cyst wall; cholesterin; scolices and hooklets absent.
Case 149.—Phelps, C., 1898. New York Medical Journal, lxvii. 218-219. Male, aged fifty-one years, New York City. Three large cysts attached to greater curvature of stomach and six to the colon; daughter cysts; no hooklets.
Case 150.—Packard, F. A., 1898. Transactions of the Philadelphia Pathological Society, xviii. 101. Female, aged thirty-four years, negro, Philadelphia, Pa. Small cyst of liver; hooklets, cholesterin, lamellated cyst wall. (Pennsylvania Hospital, 1896.)
Case 151.—Steele, J. D., 1898. Transactions of the Philadelphia Pathological Society, N. S., i. 106. Adult (Western town). Cyst of liver; scolices, hooklets, and lamellated cyst wall.
Case 152.—Mason, A. L., and Burrell, H. L., 1898. Medical and Surgical Rep., Boston City Hospital, Ser. ix. 108-115. Boston, Mass. Cyst of liver; no hooklets.
Case 153.—Mason, A. L., and Burrell, H. L., 1898. Medical and Surgical Rep., Boston City Hospital, Ser. ix. 108-115. Male, American, Boston, Mass. Large purulent cyst of liver, with secondary involvement of right pleural cavity; daughter cysts.
Case 154.—Beck, C., 1898. Journal of the American Medical Association, xxxi. 1238-1239. Male, aged thirty-eight years, Austrian (7 years in U. S.), New York City. Purulent cyst in right lung; expectorated pus containing hooklets. (St. Mark's Hospital, 1897.)
Case 155.—Beck, C., 1898. Journal of the American Medical Association, xxxi. 1238-1239. Male, aged thirty-four years, German (8 years in U. S.), New York City. Echinococcus of liver. (St. Mark's Hospital, 1892.)
Case 156.—Lane, 1899. Pacific Record of Medicine and Surgery, San Francisco, N. S., i. 314. San Rafael, Cal. Cyst of liver.
Case 157.—Berg, 1899. Medical Record, New York, lv. 26. Male, aged thirty years, New York City. Large cyst of liver; rupture into common bile-duct; passed per rectum.
Case 158.—Lilienthal, 1899. Medical Record, New York, lv. 26. Male, young adult, New York City. Cyst in left breast.
Case 159.—Willien, 1899. Journal of the American Medical Association, xxxii. 114-115. Male, aged fifty years, Fontanet, Ind. Large cyst in liver.
Case 160.—Weir, 1899.—Medical Record, New York, lv. 149-151. Male, aged thirty-nine years, German (?), New York City. Large cyst in liver; scolices.
Case 161.—Woods, G. W., 1899. Proceedings Assoc. Military Surg. of the U. S., viii. 157-160. Male, Brooklyn, N. Y. Large cyst of liver extending through diaphragm into right pleural cavity; daughter cysts, scolices, hooklets; also small cyst in head of left humerus. (U. S. Naval Hospital, Brooklyn, N. Y.)
Case 162.[3]—Cary, C., and Lyon, I. P., 1900. Transactions of the Association of American Physicians, xv. 367-379, and American Journal of the Medical Sciences, cxx. 402-413. Male, aged thirty-one years, Negro (Va.), Buffalo, N. Y. Primary exogenous cysts of right pleura; hyaline degeneration of cuticle, without gross degeneration; hooklets and cholesterin.

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[1] Sommer (New York Medical Journal, 1896, lxiv. 265) says that this case was in a Mexican, N. Y. Liver.

[2] Specimens in the museum of the University of Buffalo

[3] Ibid.

Case 163.—Henry, F. P., 1900. Philadelphia Medical Journal, v. 880, and Personal Communication. Male, aged forty-nine years, negro (Va.), Philadelphia, Pa. Two cysts in liver; hooklets.

Case 164.—Porter, C. A., 1900. Boston Medical and Surgical Journal, cxiii. 302. Male, aged seventeen years, Italian, Boston, Mass. Large cyst of liver; lamellated cyst membrane; scolices and hooklets not found. (Massachusetts General Hospital).

Case 165.—Rogers, W. K., 1900. Journal of the American Medical Association, xxxiv. 276, 277. Female, aged thirty-four years, Newark, O. Cysts of nasal septum; hooklets.

Case 166.—Odendahl, F. H., 1900. Northwestern Lancet, St. Paul, xx. 29-30. Female, aged thirty-two years, St. Paul, Minn. Large cyst of liver, containing one gallon of fluid and several hundred small cysts.

Case 167.—Odendahl, F. H., 1900. Northwestern Lancet, St. Paul, xx. 29-30. Female, aged thirty-five years, St. Paul, Minn. Liver; cysts passed per rectum.

Case 168.—Vickery, H. F., 1900. Transactions of the Association of American Physicians, xv 379., and Personal Communication. Male, aged twenty-nine years, French, Boston, Mass. Purulent mass in right pleural cavity; daughter cysts. (Mass. Gen. Hosp., 1896).

Case 169.—Shorb, J. D. B., 1900. Southern Cal. Practitioner, xv. 472-474. Female, aged fifty-eight years, German (20 years in U. S.), Los Angeles, Cal. Large degenerated cyst cavity in liver; daughter cysts; cysts passed per rectum.

Case 170.—Bartholow, 1900. Annals of Surgery, xxxii. 721-722. Male. Large single cyst on posterior surface of liver; gallon of fluid.

Case 171.—Da Costa, J. C., 1900. Annals of Surgery, xxxii. 721. Female, aged twenty-nine years. Suppurative cyst of liver.

Case 172.—Loux, H. R., 1900. Annals of Surgery, xxxii. 714-723. Male, aged thirty-one years, German, Philadelphia, Pa. Single cyst attached to lower surface of liver; weight, 197 grammes; numerous daughter cysts; hooklets, scolices, brood-capsule, cholesterin.

Case 173.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Female, aged twenty-eight years, Italian (3 years in U. S.), Brooklyn, N. Y. Single cyst arising from under surface of liver.

Cases 174-178.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Italians, Brooklyn, N. Y. Liver. (Five cases).

Case 179.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Brooklyn, N. Y. Large cyst in left lobe of liver.

Case 180.—Bolton, P. R., 1900. Annals of Surgery, xxxi. 253-254. Male, aged eighteen years, Italian (3 years in U. S.), New York City. Single cyst in under surface of liver; lamellated cyst wall; no hooklets.

Case 181.—Mercier, O. F., 1900. L'Union méd du Canada, Montreal, xxix. (N. S., v.), 427-438. Male, aged fifty-five years, Canadian, Montreal, Quebec. Large cyst in the region of the right kidney; daughter cysts. (Hôpital Notre-Dame, 1900).

Case 182.—Mercier, O. F., 1900. L'Union méd du Canada, Montreal, xxix. (N. S., v.), 427-438. Male, aged thirty-four years, Syrian, Montreal, Quebec. Large cyst of liver; daughter cysts. (Hôpital Notre-Dame, 1900).

Case 183.[1]—Park, R., 1901. Personal communication. Male, aged thirty-five years, Buffalo, N. Y. Multilocular cyst of liver, degenerated; lamellated cyst wall; no scolices or hooklets.

Case 184.[2]—Park, R., 1901. Personal communication. Male, aged twenty-six years, English (2 years in U. S.), Lyndonville, N. Y. Two cysts in liver, one calcified and one, as large as an infant's head, protruding into abdominal cavity.

Case 185.—Fronczak, F. E., 1901. Personal communication. Female, aged forty-five years, Polish, Buffalo, N. Y. Single cyst of liver; hooklets. (Erie Co. Hospital, Buffalo, 1897).

Case 186.[3]—Kenerson, V., 1901. Personal communication. Male, aged thirty-five years. Irish, New York City. Cyst of liver, purulent; daughter cysts and hooklets. (New York Hospital, 1896).

Case 187.—Packard, F. A., 1901. Personal communication. Male, negro, Philadelphia, Pa. Hydatid cyst in liver; hooklets and scolices. (Philadelphia Hospital.)

Case 188.—Packard, F. A., 1901. Personal communication. Female, aged fifty-nine years, negro, Philadelphia, Pa. Small cyst in liver; hooklets, cholesterin, and lamellated cyst wall. (Pennsylvania Hospital, 1887.)

Case 189.—Packard, F. A., 1901. Personal communication. Female, aged twenty-seven years, negro, Philadelphia, Pa. Multilocular hydatid cyst of right breast; hooklets (Pennsylvania Hospital, 1889).

Case 190.—Packard, F. A., 1901. Personal communication. Female, aged forty years, Syrian, Philadelphia, Pa. Cyst of liver. (Pennsylvania Hospital, 1889.)

Case 191.—Chown, H. H., 1901. Personal communication. Female, aged thirty-two years, Icelander, Winnipeg, Man. Neck; hooklets.

Case 192.—Chown, H. H., 1901. Personal communication. Male, aged thirty-six years, Icelander, Winnipeg, Man. Spleen; daughter cysts.

Case 193.—Chown, H. H., 1901. Personal communication. Male, aged eighteen years, Icelander, Winnipeg, Man. Arm, above elbow; hooklets.

Case 194.—Chown, H. H., 1901. Personal communication. Female, aged twenty-five years, Icelander, Winnipeg, Man. Cyst of liver, resembling a dilated gall bladder; hooklets; subsequently operated on in Boston for a second cyst.

Case 195.—Chown, H. H., 1901. Personal communication. Male, aged twenty-four years, Icelander, Winnipeg, Man. Left iliac region, apparently post-peritoneal; daughter cysts.

Case 196.—Chown, H. H., 1901. Personal communication. Male, aged fifty-six years, Icelander, Winnipeg, Man. Lung; expectorated collapsed daughter cysts for months.

Case 197.—Chown, H. H., 1901. Personal communication. Female, aged thirty-four years, Icelander, Winnipeg, Man. Pelvis; rupture into bladder; cysts passed per urethram.

Case 198.—Chown, H. H., 1901. Personal communication. Female, aged forty-six years, Icelander, Winnipeg, Man. Calcareous cyst of liver; death from intestinal hemorrhage.

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[1] Specimens in the museum of the University of Buffalo.

[2] Ibid.

[3] Specimens in the museum of the New York Hospital.

Case 199. Chown, H. H., 1901. Personal communication. Male, aged thirty-seven years Icelander, Winnipeg, Man. Liver; hooklets.

Case 200.—Chown, H. H., 1901. Personal communication. Female, aged twenty-three years, Icelander, Winnipeg, Man. Cyst of peritoneal cavity.

Case 201.—Chown, H. H., 1901. Personal communication. Female, aged seventeen years, Icelander, Winnipeg, Man. Liver; hooklets.

Case 202.—Chown, H. H., 1901. Personal communication. Male, aged twenty-seven years, Icelander, Winnipeg, Man. Cyst of liver; daughter cysts.

Case 203.—Chown, H. H., 1901. Personal communication. Female, aged twenty-three years, Canadian (both parents Icelanders), Winnipeg, Man. Cyst of liver; daughter cysts.

Case 204.—Chown, H. H., 1901. Personal communication. Female, aged twenty-six years. Icelander, Winnipeg, Man. Peritoneal cavity, cyst floating free; patient was previously operated on in London for large hydatid of left lumbar region.

Cases 205–217.—Chown, H. H., 1901. Personal communication. Thirteen cases (in addition to the above), eight in females and five in males; ages, 23, 26, 31, 33, 33, 36, 37, 39, 42, 43, 44, 45, and 51 years. Icelanders, Winnipeg, Man. All cases had cysts of the liver, with daughter cysts, and varying only in size.

Case 218.—Bangs, L. B., 1901. Annals of Surgery, xxxiii. 565–573. Male, aged thirty-nine years, German, New York City. Cyst of prostate; daughter cysts and hooklets. (Bellevue Hospital, 1900.)

Case 219.—Gay, W. F., 1901. Boston Medical and Surgical Journal, cxliv. 492–493. Male, aged twenty-five years, Italian (4 years in U. S.), Boston, Mass. Hydatid of liver, with rupture into lung and expectoration of cysts; sputum contained scolices, lamellated cyst membrane, etc.

Case 220.—Peters, G. A., 1901. Canadian Practitioner and Review, xxvi. 75–78. Male, aged twenty years, Argentine Republic, Guelph, Ont. Large cyst in tail of pancreas [clinical diagnosis of site]; brood capsules and hooklets

MUSEUM SPECIMENS[1] (NOT REPORTED).

Case 221.—McGill Medical School Museum, Montreal. Male, aged thirty-five years, Italian (4 years in America), Montreal, Quebec. Multiple cysts in liver, spleen, stomach, omentum, mesentery and pelvis; perforation of stomach and duodenum (1880).

Case 222.—McGill Medical School Museum, Montreal. Female, aged forty years, English, Montreal, Quebec. Obsolete cyst in liver; hooklets.

Case 223.—McGill Medical School Museum, Montreal. Montreal, Quebec. Cyst in liver.

Case 224.—Miner. United States Army Medical Museum, Washington, No. 8014. Male, adult, colored, Alexandria, Va. Echinococcus of brain.

Case 225.—Bond. United States Army Medical Museum, Washington, No. 8089. Male, aged fifty-one years, light mulatto, Washington, D. C. Cyst in liver.

Case 226.—Warren Anatomical Museum, Harvard, Boston, No. 2381. Cyst in liver.

Case 227.—Warren Anatomical Museum, Harvard, Boston, No. 2156. Cavity at apex of left lung, containing echinococci.

Case 228.—Warren Anatomical Museum, Harvard, Boston, No. 3773. Echinococci passed *per rectum*.

Case 229.—Warren Anatomical Museum, Harvard, Boston, No. 3871. Male, adult. Cyst in liver.

Case 230.—Bellevue Hospital Museum, New York, No. 865. Cyst in liver.

Case 231.—Bellevue Hospital Museum, New York, No. 866. Cyst in liver.

Case 232.—Bellevue Hospital Museum, New York, No. 867. Suppurating cyst of liver.

Case 233.—New York Hospital Museum, No. 932. New York City. Cyst in liver.

Case 234.—New York Hospital Museum, Nos. 933–937. New York City. Multiple echinococci: one in abdominal wall, one on surface of liver, a third loose in peritoneal cavity, and a fourth in pelvis.

Case 235.—Loomis. Museum of University of New York. Large cyst of liver.

Case 236.—Museum of Pennsylvania Hospital, Philadelphia, No. 1382[50]. Cyst in liver.

Case 237.—Museum of Pennsylvania Hospital, Philadelphia, No. 1382[55]. Male, lad, French. Multiple cysts in liver.

Case 238.—Museum of Pennsylvania Hospital, Philadelphia. Aged fifty-five years, Italian. Philadelphia, Pa. Cyst in liver.

Case 239.[2]—Museum of University of Pennsylvania, G. B. Wood Cabinet, P. C., 46, vol. i. Several cysts from liver.

Case 240.[3]—Museum of University of Pennsylvania, G. B. Wood Cabinet. Hydatid cyst of spleen

Case 241.[4]—Museum of University of Pennsylvania, G. B. Wood Cabinet. Liver with a cyst, probably hydatid.

AGE. The occurrence of the disease according to age is shown in Table II. by decades, and graphically in the accompanying age-chart:

[1] Most of these museum specimens were originally summarized by Osler (loc. cit.).

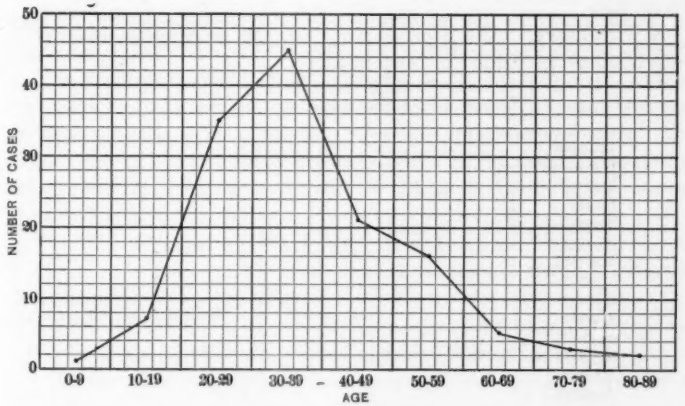
[2] Osler states that this case is perhaps not from American sources.

[3] Ibid.

[4] Ibid.

TABLE II.—AGE.

0– 9 years	1[1]		
10–19 "	7		
20–29 "	35 = 26 per ct.	59 per ct.	74 per ct.
30–39 "	45 = 33 "		
40–49 "	21 = 15 "		
50–59 "	16		
60–69 "	5		
70–79 "	3		
80–89 "	2		
	135		
Child	1		
Lad	1		
Young adult	2		
Adult	19		
Old	4		
Unstated	79		
	106		
Total	241		



A large majority (74 per cent.) of the cases whose ages were given occurred in young and middle-aged adults. In the age-period—twenty to thirty-nine years—59 per cent. of the cases occurred. Jonassen[2] found 55 per cent., and Finsen[3] 42 per cent. in Iceland in this period of life. Under the age of ten years the disease is rare, in spite of the fact that young children are apparently more exposed than adults to infection. As shown by the table, no period of life is exempt.

SEX. The cases were divided by sex, as follows:

[1] Ferguson (vide infra) saw three cases in children under eight years of age, who had been brought to Winnipeg by Icelandic emigrants. These cases were not described individually, and have therefore been omitted from this enumeration.

[2] Jonassen. Echinokokkensygdomen belyst ved Islandske Lægers Erfaring, Kjöbenhavn, 1882.

[3] Finsen. Jagttagelser angaaende sygdoms-fortsoldener, x., i. Iceland, Kjöbenhavn, 1874.

TABLE III.—SEX.

Males	104	= 60 per cent.
Females	69	= 40 "
	173	
Not stated	68	
	241	

The preponderance of males over females (3 to 2) in the American cases is about reversed in the statistics of most authorities. Neisser[1] gives 34.8 per cent. in males and 65.2 per cent. in females; Finsen[2] found 29.1 per cent. in males and 70.9 per cent. in females, and Jonassen's[3] figures show 40.6 per cent. in males and 59.4 per cent. in females. According to Davaine,[4] the occurrence of the disease in the two sexes is equal. The fact that a large part of the American cases occurred among foreign immigrants, among whom young adult males predominate, may explain in part the peculiarly high proportion of males in these cases.

NATIONALITY. The representation of the cases by nationality or race is shown in Table IV., as follows:

TABLE IV.—NATIONALITY OR RACE.

Birthplace.			
Iceland		58	
Germany		23	
Italy	18		
Sicily	1	19	
England	13		
Wales	1	14	
Ireland		3	
France		3	
Poland		2	
Russia		2	Foreign-born, 91 per cent.
Syria		2	
Argentine Republic		1	
Azores		1	
Austria		1	
Denmark		1	
Japan		1	
Mexico		1	
Sweden		1	
Foreign (not specified)		3	
		136	
America	1		
Canada	2		
Negro	6		
" (U. S.)	4	13	Native-born, 9 per cent.
Total stated		149	
White	4		
Not stated	88	92	
Total		241	

[1] Neisser. Die Echinococcen-Krankheit., Berlin, 1877.
[2] Finsen. Loc. cit. [3] Jonassen. Loc. cit.
[4] Davaine. Traite des Entozoires, Paris, 1877, second edition.

From this table it is seen that 136 cases were stated to be among foreigners; in 92 cases the nationality was not stated, and the remaining 13 cases were distributed as follows: Negro, 10; Canadian, 2; American, 1. Mention of American birth is conspicuously rare, though it may be inferred from the context of the reports of some cases that the subjects were native-born.

If the proportion of foreigners shown among the cases whose birthplace was stated be assumed to obtain also in the cases of unstated nationality, it is seen that 91 per cent. of the American cases occurred in foreigners. The negro has been assumed to be native-born, and, in fact, it was so stated in at least four instances. The disease seems to have been directly imported into America in the great majority of cases by emigrants from foreign countries. The relative immunity of Americans is seen even among the American-born children of the Icelandic emigrants of Manitoba, though among the latter the disease is very common. The disease prevails chiefly in the lower classes and particularly among the ignorant and slovenly, but not exclusively, for instances of the disease are recorded in persons of rank and culture.

The large representation of Germans in the table is explained by the large German emigration to America and the well-known fact that hydatid disease is very common in certain parts of Germany. The occurrence of nineteen cases among Italians is not remarkable considering the habits of this people, and the disease is said to be not uncommon in England.

The following facts, quoted from Ferguson,[1] of Winnipeg, are of interest in reference to the large number of cases of hydatid disease in Icelanders shown by the table:

"First, in 1874, and at different times since, the hydatid disease was brought to Manitoba by the influx of Icelandic immigrants.

"So far as I have been able to ascertain, the disease is limited to the Icelandic population and to those of them born in Iceland.

"I have met with it in three children under eight years of age, but upon inquiry it was found that they were born in Iceland.

"The total number of my cases has been twenty-seven, but this does not include several of doubtful diagnosis, in which objection was taken to aspiration or exploratory incision.

"From the reports of our hospitals, and from conversations with the city physicians, I have traced some eighteen or twenty[2] other cases treated by different medical men.

"I think that I am justified in concluding that between forty-five and fifty persons affected with the echinococcus disease have been

[1] Ferguson. Northwestern Lancet, St. Paul, 1893, vol. xiii. pp. 41–48.

[2] These 18 or 20 cases have not been included in our tables, because they were merely estimated and no details were given.

treated in Winnipeg since 1874. I feel that this number is rather below the proper reckoning."

Ferguson gave the Icelandic population of Winnipeg as 3000, and, accepting his estimate of forty-five to fifty cases of hydatid disease seen in this population, the relative frequence of occurrence is found to be about identical with that of the disease in Iceland, where, according to Jonassen, whose estimate is probably the most accurate, one out of every sixty-one persons (1.6 per cent.) is affected with hydatid disease. Ferguson's estimate, however, is probably too conservative, judging from the number of cases shown in our statistics.

It is interesting to note that Ferguson saw no cases of the disease in the Canadian-born offspring of Icelandic emigrants. Chown, however, observed one such case in a young woman, aged twenty-three years. This case, therefore, gives ground for the apprehension that an endemic focus of the disease has already been established in Winnipeg by the Icelandic colony, and that further cases may occur in the children of Icelanders as they reach maturity, the period of life in which the disease is most commonly observed.

GEOGRAPHICAL DISTRIBUTION. The geographical distribution of the American cases is shown in Table V., as follows:

TABLE V.—GEOGRAPHICAL DISTRIBUTION.

New York	59	Connecticut	1
Manitoba	56	Louisiana	1
Pennsylvania	24	Michigan	1
Massachusetts	13	New Jersey	1
Ohio	9	Tennessee	1
Missouri	8	Vermont	1
Ontario	8	Washington	1
Quebec	7		220
Texas	6		
Illinois	5	Not specified	21[1]
California	4		
Minnesota	3	Total	241
District of Columbia	3		
Alabama	2	United States	150
Indiana	2	Canada	71
Kentucky	2	Not stated	20
Virginia	2		241

There seems to be nothing noteworthy in the geographical distribution shown, except the fact that concentration of the disease in certain States is coincident with concentration of population, and especially foreign immigrant population in such States. Attention has already been called to the prevalence of the disease among the Icelandic immigrants of Manitoba. It is noticeable that so few cases are furnished by the great grazing and cattle-raising States of the West, where it would

[1] Includes one case from a "Western town" in the United States.

seem, *a priori*, that the conditions favorable to the spread of the disease might prevail.

ANATOMICAL LOCATION. The distribution of the disease by organs in the body is shown in Table VI., as follows:

TABLE VI.—ANATOMICAL LOCATION.

Liver			177	= 73.7 per cent.
Omentum	8			
Peritoneal cavity	8			
Peritoneum	5		26	= 10.8 "
Mesentery	5			
Lung			11	= 4.5 "
Spleen			9	= 3.7 "
Kidney			9	= 3.7 "
Bladder			8	= 3.3 "
Pelvis	5			
Walls of pelvis	1		7	= 2.9 "
Connective tissue of pelvis	1			
Pleura			5	= 2.0 "
Brain			4	= 1.6 "
Breast			4	= 1.6 "
Abdominal wall			4	= 1.6 "
Stomach			2	= 0.8 "
Common bile-duct			2	= 0.8 "
Neck			2	= 0.8 "
Arm			1	= 0.4 "
Humerus			1	= 0.4 "
Pancreas			1	= 0.4 "
Eye ("bulbus of")			1	= 0.4 "
Diaphragm			1	= 0.4 "
Sub-diaphragmatic			1	= 0.4 "
Intestine			1	= 0.4 "
Muscles of back			1	= 0.4 "
Ovary			1	= 0.4 "
Pericardium			1	= 0.4 "
Prostate			1	= 0.4 "
Sac of inguinal hernia			1	= 0.4 "
Testicle			1	= 0.4 "
Uterus			1	= 0.4 "
Psoas muscle			1	= 0.4 "
Muscles of thigh			1	= 0.4 "
Nasal septum			1	= 0.4 "
			287 organs in 240 cases.	
Not stated				1 case.
				241 cases.
Cysts passed per rectum		10		
Cyst membrane passed per rectum		1	11	= 4.5 per cent.
Cysts expectorated		6		
Hooklets expectorated		1	7	= 2.9 "
Cysts vomited		3		
Cyst membrane vomited		1	4	= 1.6 "
Cysts passed per urethram		2	2	= 0.8 "

The liver is seen to be the seat of election of the parasite, being involved in 73.7 per cent. of the cases—a high rate compared with other statistics. Davaine[1] gives for the liver 44 per cent.; Neisser[2] 50

[1] Davaine. Loc. cit.

[2] Neisser. Loc. cit.

per cent., and Finsen[1] 69 per cent. Next to the liver the order of frequence of the organs involved by the disease is as follows: omentum, peritoneal cavity, peritoneum, and mesentery (combined), 10.8 per cent.; lung, 4.5 per cent.; spleen and kidney, each, 3.7 per cent.; bladder, 3.3 per cent., etc. Very unusual sites of the parasite are shown in our statistics in the sac of an inguinal hernia, nasal septum, pericardium, head of the humerus, brain, eye, prostate, testicle, pancreas, and pleura (primary), in one case each. The multilocular type of cysts (*echinococcus multilocularis*) is mentioned in four cases—three times in the liver and once in the female breast. Two of these cases were in negresses, in the breast and in the liver. Rupture of cysts into the following organs is mentioned: intestine, stomach, bile-ducts, bladder, peritoneal cavity, and lung. Rupture through the chest wall externally took place in one case. Discharge of cysts, cyst membrane, or hooklets by the various natural outlets of the body was mentioned as follows: Passed per rectum, 11 cases (4.5 per cent.); expectorated, 7 cases (2.9 per cent.); vomited, 4 cases (1.6 per cent.), and passed per urethram, 2 cases (0.8 per cent.).

Diagnosis. The diagnosis of echinococcus disease in the American cases seems to have been based on the gross appearances of the cysts, the presence of daughter cysts, the site of the cysts, etc., in a majority of the cases. However, in a fair proportion of the more recently reported cases, such characteristic microscopical elements as brood capsules, scolices, hooklets, calcareous corpuscles, lamellated cyst membrane, etc., were mentioned in support of the diagnosis. The chemical tests of the cyst fluid for the absence of albumin and the presence of grape-sugar were commonly applied, and the specific gravity was tested in many cases. Succinic acid, said by Leuckart to be "found in hardly any other living organism," and pathognomonic of hydatid fluid, was looked for in only a few cases. The physical characteristic of the outward curling of the elastic cyst wall, when incised, was only rarely mentioned. In doubtful cases none of these points of diagnosis should be neglected, though any or all of them may be wanting in atypical or degenerated cysts.[2]

It has been a difficult matter, in reviewing the literature, to decide what cases ought to be excluded because of insufficient evidence of the true cestode nature of the cysts, and perhaps an occasional case has been included in the summary of cases that might better have been omitted. Without being hypercritical, we have tried to be conservative in judging of cases. It is possible, also, that an occasional case

[1] Finsen. Loc. cit.

[2] Cary and Lyon. The American Journal of the Medical Sciences, October, 1900, vol. cxx. pp. 402–413; and Transactions of the Association of American Physicians, 1900, vol. xv. pp. 367–379.

may have been reported independently by different observers, and thus included more than once in our summary, though we have been on our guard against this source of error.

Is the Disease Increasing? This important question, we believe, cannot be answered authoritatively at present. It is true that the great majority of cases in our statistics are accredited to recent years. This fact, however, cannot be accepted as evidence that the disease is more common now than formerly. It is only within recent years that the habit of reporting rare and interesting cases in the medical journals has become general. Probably more cases of pneumonia, typhoid fever, or any other disease that might be mentioned, have been reported in the medical literature in the last quarter of a century than in all previous time. This fact plainly does not argue that these diseases are increasing in prevalence, but merely that they are being better recognized, studied, and discussed than formerly. So we are not able to affirm that hydatid disease is increasing in America. Still, one of the best authorities, Dr. C. W. Stiles,[1] zoölogist of the Bureau of Animal Industry, has stated that the disease in the domesticated animals in the United States is undoubtedly on the increase. If this be true, it is probably true also that the disease in man is on the increase in this country. Probably only a small portion of the cases occurring in the country are recognized and reported. However, the disease is still a rarity, and is considered a curiosity in medical circles.

Hydatids in Animals. Table VII. shows what is at present known of the occurrence of echinococcus cysts in animals[2] in the United States and Canada. With the exception of the records of the United States meat-inspection service, published by Dr. D. E. Salmon, chief of the Bureau of Animal Industry, the evidence seems to indicate that hydatid cysts in our domesticated animals are not uncommon, different observers finding them in from 1 to 10 per cent. of animals examined. It is difficult to reconcile these various observations with the enormous statistics of the government meat-inspection service, which indicate that the disease is a great rarity in the live-stock of the United States. In

[1] Stiles, C. W. Bull. No. 19, United States Department of Agriculture, Bureau of Anima Industry, Washington, 1898, p 122.

Doctor Stiles, in a personal communication, writes as follows: "It is largely on the base of our unpublished statistics that I have insisted so frequently upon the necessity of guarding against hydatid disease, which is undoubtedly on the increase among the domesticated animals."

[2] The cysts described in the muscles of prairie jack-rabbits by Herff (Texas Medical Journal, 1894, vol. ix. pp. 613–616) and by Menger (ibid., 1899, vol. xv. pp. 237–45; and ibid., 1900, vol. xvi. pp. 415–20; and American Journal of Dermatology and Genito-urinary Diseases, St. Louis, 1900, vol. iv. pp. 155–163), although containing booklets, cannot be accepted as *Echinococcus polymorphus*. They were probably *Cœnurus serialis*, the larval cysts of *Tænia serialis*, a well-known bladder-worm of rabbits in this country. The writer has occasionally encountered these cysts in white rabbits, in Buffalo, in making autopsies on experimental rabbits dead of various infections.

the two years ending June 30, 1899, only six out of 8,831,927 cattle were condemned in whole or in part on account of hydatid disease, or one in 1,471,987; 209 out of 11,110,776 sheep, or one in 53,161, and 1994 out of 44,841,779 hogs, or one in 22,488. The official authority of these figures, as well as their enormous scope, support them against the evidence of the other scattered observations recorded in Table VII., and we are, therefore, bound to assume that at present echinococcus disease is a very rare affection in the native herds and flocks of this country, as it is also among the native population.

TABLE VII.—ECHINOCOCCUS IN ANIMALS IN NORTH AMERICA.

Gross, S. D., 1845. Pathological Anatomy, 2d ed., 118. Hogs. Ohio, Indiana, and Kentucky. "In Cincinnati, where there are annually slaughtered upward of two hundred thousand hogs, probably not a tenth part are free from this disease. Whole droves, consisting of three or four hundred head, are sometimes thus affected. These animals, most of which are young, are raised in the prairie districts of Ohio, Indiana, and Kentucky."

Osler, W., 1882. American Journal of the Medical Sciences, lxxxiv. 475–480. Montreal, Can. "In casual visits to butcher stalls and to the shambles I have obtained six or eight large echinococci."

Osler, W., 1882. American Journal of the Medical Sciences, lxxxiv. 475–480. Cat. Montreal, Can. Liver of a cat with two large cysts.

Dean, 1882. American Journal of the Medical Sciences, lxxxiv. 475–480. Hogs. St. Louis, Mo. "Considerable proportion of the hogs slaughtered at St. Louis are infected."

Osler and Clement, 1883. Canada Medical and Surgical Journal, xi. 325–326. Hogs. Montreal, Canada. "In the 1037 hogs examined, echinococci were found in the livers of 31, or 1 in 33.4."

Day.[1] Bureau of Animal Industry Records, Washington (unpublished). Pigs. St. Louis, Mo.

Moore.[2] Bureau of Animal Industry Records, Washington (unpublished). Washington, D. C. Hydatids, 2 cases.

Wheeler.[3] Bureau of Animal Industry Records, Washington (unpublished). Hogs. New Orleans, La. Livers of 117 out of 2000.

Buckingham.[4] Bureau of Animal Industry Records, Washington (unpublished). Camel. Washington, D. C. Liver.

Welch.[5] Hogs. Maryland. Hydatids, 3 cases.

Stiles, C. W., 1898. Bureau of Animal Industry, Washington, D. C. Cattle, hogs, and camel. District of Columbia, Missouri, and Nebraska. "I have seen cases of hydatids in this country in cattle, hogs, the camel, and man, but as yet have seen no cases in sheep."

Stiles, C. W., 1900. Private communication. Hogs, Kansas City, Mo., and Ottumna, Iowa; Cattle, Chicago. "About 1 per cent. of the hogs I examined at Kansas City some five years ago contained echinococci. I have also found it in cattle at the Chicago abattoirs, and in hogs at the Ottumna (Iowa) abattoir, besides the cases published in Bull. 19 and by Sommer."

Salmon, D. E., 1901. Journal of the American Medical Association, xxxvi. 867–871. Sheep. United States. Hydatids ("echinococci") found in 209 out of 11,110,776 slaughtered sheep inspected by the United States meat inspection service during the two years ending June 30, 1899.

Salmon, D. E., 1901. Journal of the American Medical Association, xxxvi. 867–871. Cattle. United States. Hydatids found in 6 out of 8,831,927 slaughtered cattle inspected by the United States meat inspection service during the two years ending June 30, 1899.

Salmon, D. E., 1901. Journal of the American Medical Association, xxxvi. 867–871. Hogs. United States. Hydatids found in 1994 slaughtered hogs inspected by the United States meat inspection service during the two years ending June 30, 1899.

The United States meat-inspection service, however, gives only the aggregate for the whole country, and furnishes no facts on the special distribution of the disease in different parts of the country. It is quite likely that the disease is not evenly distributed throughout the country, but prevails in certain districts in greater proportion than in other districts, and that endemic foci of the disease exist, perhaps, here and there. Such a presumption is favored by the known facts of the distribution of the disease in Germany and other countries, and would help to explain the greater frequence of the disease in animals reported from certain parts of the country by various individual observers.

[1] Cited by Sommer, New York Medical Journal, 1895, lxii. 656–659.
[2] Ibid. [3] Ibid. [4] Ibid. [5] Ibid.

Occurrence of the Adult Tapeworm (Tænia Echinococcus) in Dogs. It would, therefore, appear, *a priori*, that the adult *Tænia echinococcus*, from whose eggs when introduced into the gastro-intestinal tract are developed the larval hydatid cysts, must be an exceedingly rare tapeworm in the dogs of North America. And this is shown to be the case by the fact that this tapeworm in dogs has been discovered and confirmed by competent authority[1] in only a single instance in the United States, by Curtice[2] in Washington, D. C. Osler[3] failed to find it in "some scores of dogs" examined in Canada from 1867 to 1882. Osler and Clement,[4] in 1883, from Montreal, wrote: "We have never met with a specimen in numerous dissections." Sommer[5] examined fifty dogs in Washington, D. C., in 1896, without finding it. Ward[6] did not discover it among twenty dogs examined in Lincoln, Neb., in 1897. Stiles[7] and Hassall,[8] of the Bureau of Animal Industry, Washington, D. C., have never in their large experience seen it from any American source, except the single specimen discovered by Curtice. Sommer,[9] in 1900, wrote: "In numerous dogs examined since 1896 at Blackwell's Island, New York, and in the State of New York I have never succeeded in finding the *Tænia echinococcus*." More extensive investigations than heretofore made, however, are required for determining the distribution and rate of occurrence of this parasite in dogs on this Continent. The minute size of the tapeworm makes its detection difficult, and throws the burden of investigating its occurrence on the few scientific zoölogists, veterinarians, and physicians who have given special attention to the much-neglected study of helminthology.

Prophylaxis. Echinococcus disease in man and animals is one and the same disease, derived from a common source, namely, the ingestion with food or drink of the eggs of the *Tænia echinococcus*—a tiny tapeworm inhabiting the intestinal tract of dogs and wolves. The dog or wolf represents the determinate host of the parasite, and man and various animals the intermediary host in which the larval or bladder stage of the parasite in its cycle of development is undergone. For human beings or animals to become infected with the larval stage or hydatid cysts, they must in some way receive the eggs of the adult tapeworm of dogs into their stomachs, where the capsule or shell of

[1] Herf (Texas Medical Journal, 1894, vol. ix. pp. 613–616), who claims to have found the *Tænia echinococcus* in his district (San Antonio, Texas) in all dogs dissected, was probably dealing with some other variety of tapeworm, as already suggested by Sommer (loc. cit.).

[2] Curtice. Records of the Bureau of Animal Industry, Washington, D. C.

[3] Osler. The American Journal of the Medical Sciences, 1882, vol. lxxxiv. p. 475.

[4] Osler and Clement. Canada Medical and Surgical Journal, 1883, vol. xi. p. 325.

[5] Sommer. Veterinary Magazine, August, 1896.

[6] Ward. Report of Nebraska State Board of Agriculture for 1896, p. 173.

[7] Stiles. Personal communication.

[8] Hassall. Bull. No. 19, United States Department of Agriculture, Bureau of Animal Industry, Washington, D. C., 1898.

[9] Sommer. Personal communication.

the egg is digested and the embryo is released and enabled to penetrate the wall of the stomach or intestine, and be carried to its destination in the liver or elsewhere in the body, where it undergoes its metamorphosis into a bladder or hydatid cyst. For dogs to become infected with the adult tapeworm it is necessary that they ingest the live scolices contained in hydatid cysts; in other words, that they eat the cysts or cyst contents of animals infected with the disease. The full cycle of development in the life-history of the parasite is thus established.

This leads us to a consideration of the means of controlling the spread of the disease, which can be effected in two ways, viz., first, by carefully destroying, by burning, the larval cysts in slaughtered animals and thus preventing the infection of dogs; and, second, by guarding ourselves against infection from dogs by "recalling that the dog is not a human being and should not be treated as one. Too intimate association with dogs is sure to breed the disease in man." (Stiles.) Stray and ownerless dogs should be killed. But, though we can in a large measure protect ourselves against infection from dogs, we cannot equally guard our domesticated animals against such infection, except by first protecting our dogs against infection from diseased cattle, sheep, and swine. And this is the *key to the whole problem. We can exterminate the disease by enforcing proper sanitary regulations at the slaughter-houses.* Compel the destruction by burning of hydatid cysts found in slaughtered animals, and the infection of dogs and the subsequent infection of man or animals from dogs are prevented, except by remote possibilities hardly worthy of consideration. The practice of throwing out the offal of slaughtered animals should be interdicted by law, and dogs should be prohibited from entering the premises of slaughter-houses. The worst offender against these plain rules of public hygiene is the *country slaughter-house*, and against it our first and chief efforts for reform must be directed, as already insisted upon by Stiles.[1]

"Hydatid disease is at present comparatively rare in this country, and now is the time to attack it. By proper precautions at the abattoirs and slaughter-houses this dangerous parasite can be totally eradicated from the country. If these precautions are not carried out it will be only a question of time when this country will take its place with Germany and Australia in respect to the number of human lives sacrificed to a disease which has not yet gained much ground with us and can now be easily controlled."[2]

Commercial Considerations. Finally, it may not be amiss to mention the possible commercial importance that this disease in our

[1] Stiles, C. W. The Country Slaughter-house a Factor in the Spread of Disease. Year Book of the Department of Agriculture for 1896, pp. 155–166.

[2] Stiles, C. W. Bull. No. 19, United States Department of Agriculture, Bureau of Animal Industry, Washington, D. C., 1898, p. 4.

live-stock may in the future assume if it is now neglected and allowed to gain a permanent place on this Continent. In view of our past experiences in the matter of international commercial jealousies and reprisals against American cattle by certain European nations, under the pretense of sanitary safeguards, the United States Government may well give serious heed to the possibility of furnishing to countries commercially hostile to us a plausible pretext for the exclusion of American animals, if by its present negligence it allows hydatid disease to become endemic and widespread in this country, as it is in Germany, Australia, the Argentine Republic, and many other parts of the civilized world.

In conclusion, we wish to acknowledge our indebtedness for notes of unpublished cases to various gentlemen mentioned in the summary of cases, and, in particular, to Dr. H. H. Chown, of Winnipeg, for his private notes of a large number of cases in Icelanders, and to Dr. Frederick A. Packard, of Philadelphia, for his generosity in placing at our disposal his references to cases which he had been systematically collecting from the literature with a view to ultimate publication.

RECENT VIEWS OF THE ORIGIN AND NATURE OF HERPES ZOSTER.

By Arthur Van Harlingen, M.D.,
of Philadelphia.

Some years ago my friend Dr. Curtin called my attention to some cases observed by him, which seemed to point to the occurrence of herpes zoster on the pleura, the peritoneum, and in the articulations. He asked me whether I knew of any similar cases. I had not had any personal experience, but determined to examine the literature of herpes zoster with this point in view. It is only recently that I have had the opportunity of doing this, but, although references to the subject are scanty, quite a number of cases have been published which go to confirm Dr. Curtin's observations.

At the same time I have found so much of interest in scattered monographs of recent date that I have thought well to give some account of the views generally prevalent among dermatologists and neurologists regarding the nature of herpes zoster.[1] In order to do this satisfactorily, it seems desirable to give a hasty sketch of what is known regarding the pathology of the disease and of the various theories as to its origin.

[1] Dr. Curtin's views are stated in several monographs. "Is Herpes Zoster a Cause of Pleurisy and Peritonitis?" Sanitarian, December, 1890, and in other publications. Some valuable observations will be found in his latest contribution to the subject, to appear in the next number of the Journal.

Barth, in an account of herpes zoster, or, as I shall call it for convenience, zoster, says that Parrot, in 1857, advanced the theory of zoster as a secondary disease, subordinate to a neuralgia, and Charcot, in 1859, called attention to a case, the first recorded, where zoster followed an injury. In 1861 Bärensprung, in his classical paper, developed the idea of the nervous origin of zoster, and attributed it to a disease of the ganglionic system, an irritation of the spinal ganglia, or of the Gasserian ganglia, while admitting that peripheral irritation of a nerve might result in a limited outbreak of the eruption.

In 1864 Mitchell, Morehouse, and Keen showed that irritation and not section of a nerve was the cause of zoster and similar eruptions. Later it was shown that compression by tumors, etc., might result in zoster on the distribution of the implicated nerve.

In 1872 Charcot reported a case of double zoster of the trunk following acute myelitis of the anterior cornua, and later reported cases of zoster occurring in connection with locomotor ataxia.

The mode of action of the morbid influence through the nerve on the skin was early made the subject of speculation, and several theories have been brought forward. What was known as the *vasomotor theory* assumed that the contraction, expansion, or paralysis of bloodvessels, and the consequent increase or decrease in the amount of nourishment brought to the cells, might lead to nutritive changes. This failed of experimental proof.

Samuel suggested a theory of *trophic nerve fibres* which was ingenious, but the existence of such fibres never having been proved, the theory fell to the ground.

The theory of the *trophic action of sensory nerves* attributes to the ordinary sensory and motor nerve fibres those functions for which Samuel had provided a special group of nerves. The nervous system acts on the nutrition of the anatomical elements by maintaining their functional activity. Trophic influence is thus transmitted to the skin and mucous membranes by the sensory nerves. Any alteration in the sensory fibres would result in the enfeeblement of the trophic influence of spinal and Gasserian ganglia upon these fibres and probably through their mediation on certain elements of the skin itself.

If this is the case we must admit that the weakening of this trophic influence would create a state of least resistance, or of greater vulnerability in those parts of the skin connected with the peripheral extremities of the affected sensory nerves.

Under these conditions, an irritant local cause, such as pressure, rubbing of the skin by clothing, or even contact of the air, might determine the appearance of the herpetic eruption, and, as would appear from recent observations, a microbic invasion could also occur.

Although this theory is attractive, and, if well founded, would

explain the pathology of zoster better than any yet brought forward, it has not as yet been proved, and remains a theory like the others.

The simplest and most plausible theory of the production of zoster, and one, indeed, which must account for a certain number of cases, is that of *propagated neuritis*. This supposes that zoster is simply the result of an inflammatory irritation transmitted by continuity of tissue from a nerve twig, or branch, to the cutaneous elements among which it is distributed.

I may say here in passing, that these theories take for granted that all zosteriform eruptions are of the same nature, and that each belongs to a specific affection: herpes zoster. In the present state of our knowledge, however, this is by no means universally admitted. We may have a specific herpes zoster, as we believe there is a specific pemphigus, while at the same time we may have various zosteroid eruptions, as we have various bullar or pemphigoid eruptions, which may arise from various causes.

Such, at least, is the generally prevalent belief among the dermatologists of to-day.

At the time Barth wrote, discussions upon the etiology and pathology of zoster seemed premature. Zoster was to be recognized as the cutaneous expression of a nervous disturbance, irritative in character, affecting, sometimes the peripheral sensory nerves, and sometimes the spinal and cranial ganglia. Sometimes resulting from an injury, as contusion or compression of a nerve, sometimes as the result of cold, or under the influence of a general perturbation of the system.

The nerve disturbance might at times be so slight as not to affect perceptibly the anatomical integrity or the functions of the nerve fibres, and might show itself only by the cutaneous eruption.

At other times, however, and especially in the old, this slight initial disturbance might prelude a destructive neuritis, which might give rise to anæsthesia, rebellious neuralgias, and various other disorders of sensation so common as the result of severe zoster.

Although, most commonly, the morbid process produces only slight skin lesions, the irritation might extend to other organs innervated by the same nerves, and might determine grave and irreparable disorders. Ulceration of the cornea, as observed by Hutchinson and Hybord many years ago, and which is an occasional accompaniment of ophthalmic zoster, was one of the first of these complications to attract attention. Jouffroy pointed out that the neuritis of zoster may be propagated to neighboring nerves, and might cause atrophy of certain muscles. Such was the position of the zoster question as stated by Barth in his article, to which reference has been made. He hazarded, however, the suggestion that visceral organs might be attacked under the influence of the same cause as that producing the zoster in a given case, and referred

to the opinion of Fernet, that certain pneumonias may be the expression of a sort of zoster of the pneumogastric nerve.

This prophesy, put forth by Barth twenty years ago, has been verified by subsequent clinical observations. A large number of cases of visceral disorders of various kinds in connection with zoster have been reported of late years. Pitres and Vaillard, Leroux, Potain, Huchard, Chandeleux, Mongour and Michel, Cantrell, Curtin, and others have shown the concurrence of zoster with tuberculosis, pleurisy, peritonitis, etc. Palm reported the case of an infant with bilateral zoster of the third branch of the trigeminal, which developed on the third day of a follicular angina. Hervonet reported a case of cervicobrachial zoster, accompanied by paresis of the arm, and, in addition, incomplete facial paralysis and ptosis. The various symptoms occurred simultaneously and, as will be observed, in different nerve tracts.

While these and similar cases continued to appear, other observations were published, going to show a close connection between zoster and various infective diseases. Gaucher and Barbe wrote on zoniform syphilides, Julien on zona and syphilis, and Spillman and Etienne published observations of a similar character. In the cases reported by the first of these writers, the distribution, as I understand, rather than the character of the lesions, lead to its denomination of zoniform, but the point of interest lies in the suggestion of the authors that the lesions had been produced, in all probability, under the influence of the pathogenic agent of syphilis or of its toxin upon the spinal axis.

Cancer, otherwise than through the direct influence of pressure from the new growth, has been suggested as a cause of zoster. Carrière, in a case of femoro-cutaneous zoster following cancer of the uterus, found parenchymatous neuritis of the nerve involved. There was no cancerous infiltration of the nerve, and Carrière attributed the neuritis to the local and direct action of cancer toxines. One of Dr. Curtin's cases was also connected with cancer of the œsophagus.

The occurrence of zoster in connection with coal-gas poisoning and as a result of the administration of arsenic presents certain problems which have not been studied with sufficient care to enable us to express any opinion. The fact that zoster or zosteriform eruptions may occur under such circumstances is established, but more cannot be said.

Wasilewsky, in his monograph on zoster as an infectious disease, cites reports of epidemics of the affection. He regards the question of immunity as settled, but with the numerous cases of recurrent zoster reported of late years, it is difficult to agree with him on this question, unless, indeed, we come to recognize the recurrent cases as zosteroid in character, rather than as true zoster.

In typical cases, prodromata, as gastric disturbance, and fever appearing several days before the eruption, with a temperature often reaching

104° F., and ceasing with the outbreak of the eruption, point, in Wasilewsky's opinion, to an infectious origin for the disease, and this view is supported by the observations of Head and Campbell, which I shall cite later.

A point in favor of the infectious character of zoster is the occurrence of enlarged lymphatic glands. Grindon and Hay have shown that true zoster is always preceded by adenopathy in the neighborhood of the eruption, or even by generalized involvement of the lymphatic glands. Hay discovered cocci in the glands.

The infectious nature of zoster having been so strongly indicated by the observations of numerous writers, it is not surprising that facts should now begin to appear in favor of its contagious character. Ferré has reported some very interesting cases in this connection. He thinks that in some cases zoster may be the consequence of a meningeal or spinal affection, and in the epidemic form may be an indication (*symptome révélateur*) of an attenuated form of cerebro-spinal meningitis. As numerous instances of the recurrence of zoster have been reported of late years, these must be taken into consideration before the infective theory is generally accepted. The view that there are several varieties of zoster, or that there is one specific zoster, an infective disease, and, therefore, not recurrent, and various zosteroid eruptions due to different causes, is one that seems to have gained considerable adherence of late years, and in the light of recent observations is certainly a plausible explanation of numerous otherwise anomalous phenomena.

The anatomy of the lesions of zoster themselves has been made the subject of investigation in the light of more recent knowledge and with the aid of modern methods. Peffer maintains that the true cause of zoster is in all cases a protozoid, which he has found in the affected skin and has described. Hartzel, who has made similar investigations, says that these protozoids are not present in ordinary inflammations of the skin, but are peculiar to zoster, variola, vaccinia, and varicella. Gilchrist, after an elaborate investigation of the subject, concludes, however, that the so-called protozoids are really the nuclei of epithelial cells.

Up to within the past year or two, Bärensprung's observations and conclusions have never been questioned, and zoster has everywhere been received as an affection closely connected with the sensory nerves, and following in its occurrence the distribution of these nerves in the skin.

It cannot have failed, however, to strike the careful observer that the eruption of zoster does not invariably follow the line of distribution of the nerves, this circumstance being particularly noticeable in thoracic zoster. Brissaud, a recent writer, goes so far as to assert that in a large number of cases the concordance of the eruption of zoster with a nerve path is a fiction.

Having made the same observation with regard to other diseases besides zoster, Brissaud sought for an explanation and found it in the theory of "metamerism."

Concluding from our knowledge of embryology that the spinal cord is composed of a series of segments superposed and relatively independent (metamers), Brissaud attempted to prove that the eruption of zoster should appear in the territory of a region innervated by a medullary metamer. This territory of innervation, however, is sometimes entirely different from that which corresponds simply to the course and distribution of the sensory nerve fibres.

Another and quite different theory of the relation of the zoster eruption to the nervous system has been proposed by Abadie. He maintains that the skin affection does not result from an alteration of the peripheral sensitive nerve fibres, nor from a medullary lesion, but from a pathological condition of the arteries and of the vasomotor nerves which regulate their dilatation in the region where the eruption is situated.

In a general way the sensory nerves accompany the arteries, at least during a portion of their course, and, therefore, the zoster eruption, which really occupies, the territory of vascular distribution, has heretofore been supposed to occupy the nerve territory. As, however, in certain localities, these two territories are neither identical nor superposable, apparent anomalies supervene, anomalies which Brissaud tries to explain by his "metameric" theory above mentioned. This hypothesis is not necessary, however, to explain the topographical anomalies of the eruption, as the cutaneous lesions occur along the bloodvessels and not along the nerve filaments.

Taking ophthalmic zoster as an example, Abadie asks how it is, if zoster is due to inflammation of the trigeminal nerve, that it only occurs (save in the very rarest cases) in the first or ophthalmic branch. As a fact, maintains Abadie, the eruption extends over the area supplied by the *supra-orbital*, *frontal*, and sometimes the *nasal* arteries.

The lesions of zoster, says Abadie, are due to a trophic influence proceeding from the *sympathetic nerve fibres*, which, in the case of ophthalmic zoster, follow the course of the arteries just mentioned. The pain and other nervous symptoms, which may or may not accompany the eruptions, are due to some involvement of the sensory nerves, which may be implicated in one case and may not in another.

This theory is supported by the form of the eruption in thoracic zoster. This does not occur above the line of the third intercostal space. The arteries supplying the three upper intercostal regions, instead of proceeding from the aorta, are derived from the subclavian artery, and consequently their vascular origin and that of the vasomotors which accompany them are different from the others which come directly from the aorta.

This theory of Abadie's makes clear, so he says, the apparent anomalies of the eruption which are inexplicable otherwise, but it takes away from the sensitive nerves of relation a part which does not belong to them and assigns it to the great sympathetic. It proves that the latter really commands and regulates nutrition in all the regions to which it is distributed, whether visceral or cutaneous.

We may thus understand, also, that certain forms of ophthalmic zoster may present particularly grave cerebral complications, and may be accompanied by hemiplegia, as Brissaud has shown, in some cases. The mechanism of these lesions has been difficult to explain by the nerve theory, but it is easily understood by accepting the excessive vasomotor dilatation of certain cerebral arterioles which may provoke rupture in their territories of irrigation.

Finally, Abadie says that quinine is a specific in ophthalmic zoster. Now, quinine is essentially a vasoconstrictor medicine acting on the sympathetic and producing a constriction of the arterial vessels.

These views of Abadie have not been generally accepted, although there is certainly something in them.

But, as Hollopeau says, the character of the concomitant and consecutive pains appear necessarily to imply a participation of the sensory nerves, and also the localizations can only be explained by a metameric action.

The blood examinations which have been made in zoster are worthy of mention, although their significance is not apparent. Like the microscopical examinations of the lesions, they are materials of possible future structures, but have not yet been fitted into their place.

Leredde says the white globules may fall to 3500. The polynuclears are diminished to 45 per cent. on an average; they may fall to 37 per cent. On the tenth to the twelfth day an eosinophilia of 6 to 8 per cent. has been noted.

Head and Campbell, in a recent and extremely elaborate paper, give a large number of facts regarding the origin and distribution of zoster. These investigators have been able to map out the affected areas in zoster, and have subsequently examined the spinal cord and nerves implicated. The cases in which they have been able to make post-mortem examinations number more than the sum total of those previously recorded by all other observers.

Head and Campbell find inflammatory and destructive changes in the posterior ganglia of the cord and in the Gasserian ganglion, and degeneration in the nerves leading to the skin. In mild cases the effects of these changes gradually pass away, but in severe cases the nerve fibres that have degenerated are replaced by fibrous tissue, and whole bundles of the nerve may be sclerosed.

Degeneration in the spinal cord occurs secondary to the herpetic lesion of the ganglion.

It may be noted, referring to the brain symptoms in connection with ophthalmic zoster, that the third division of the trigeminal is connected with cells in the Gasserian ganglion which send fibres centralward to lie in the so-called ascending or spinal roots.

Head and Campbell conclude that some agent, the nature of which we are ignorant, chooses the substance of the posterior root ganglion for its selected activity, producing profound inflammatory changes. These destructive changes in the ganglion give rise to irritation of its nerve elements, and to this irritation is due the eruption on the skin.

Acute inflammation of a posterior root ganglion, due to its implication in some local inflammatory process, can apparently produce an herpetic eruption indistinguishable from an eruption which arises as a manifestation of the acute specific disease (as, for instance, a case of zoster reported by Head and Campbell, where lymphosarcoma invaded the posterior root ganglion directly).

Zoster may appear in the course of gross organic disease of the spinal cord and roots. In some such cases Head thinks the original disease simply predisposes to an attack by the specific agent supposed to be the chief etiological factor in the production of the zoster.

Head has traced nerve twigs up to the portion of skin excised for examination, and found they showed marked signs of degeneration. He did not succeed in demonstrating degenerated fibres in the actual portion of skin examined.

A typical attack of zoster arises without any obvious peripheral or cerebral cause, and, as has been said, must be considered an acute specific disease of the nervous system, for it starts with a prodromal period of varying length, during which the temperature is raised. During this period the patient feels ill and has more or less pain.

If a child, he may be supposed to be sickening from one of the ordinary specific diseases. Then the rash appears, the febrile period lasting three to five days.

Head's experience with recurrent zoster is worth noting. He met with but four cases of relapse in four hundred of zoster.

In connection with the occurrence of zoster as a specific infectious disease we must consider the large number of zoster-like eruptions evidently due to various causes, as injuries, poisoning by arsenic, coal gas, etc. All these must be ruled out from the division of true zoster.

On the other hand, the occurrence of zoster as a specific fever running a regular course and characterized by a typical eruption, prepares the mind for the reception of Curtin's theory as to the involvement of various membranes and viscera.

If the cause of zoster lies in a disorganization of certain nerve cells or nerve fibres by the toxin of a specific germ, or by an unknown

agent of any kind, there is some reason for admitting the possibility that the same agent may give rise to inflammatory symptoms in the pleura or in the articular cavities.

As Osler and others have described the various visceral complications of erythema multiforme, so we may regard the cases observed by Curtin as those of visceral complications of herpes zoster, and I have no doubt that when the attention of observers has been drawn to this aspect of the case we shall find numerous cases of such complications reported.

Conclusions. From what has preceded, the following conclusions may be drawn:

1. Under the designation zoster or herpes zoster is to be understood a specific infectious and possibly contagious exanthem, characterized in its invasion by lassitude, general malaise, chills, increased temperature, and more or less digestive disturbance.

Following this, in most cases, neuralgic pains develop along certain nerve-paths or metameric areas, together with the development of enlarged lymphatic glands.

After a period of several days, more or less, during which the symptoms mentioned, or some of them, have manifested themselves, the characteristic exanthem shows itself, and runs through a fixed cycle of development, acme, and decrudescence. The general symptoms, particularly the neuralgic pains, may continue during this period, or, in many cases, may diminish, usually disappearing with the eruption. In other instances, and particularly in older persons, the neuralgia may form a prolonged succedaneum to the regular course of the disease.

In a certain number of cases, probably, however, rare, various visceral complications may accompany the affection. Such are paralysis of sensory or motor nerves, inflammations of the pleura, peritoneum, articulations, or viscera.

2. The infection attacks chiefly the posterior (sensory) ganglia of the cord and the Gasserian ganglion. From thence inflammation and degeneration may extend along the nerve trunks and fibres.

No other lesions have as yet been discovered post-mortem, although it is probable that further observation will result in tracing the disease in the various membranes and viscera when its presence has been clinically noted.

The numerous examinations of the skin lesions and such blood examinations as have been made have not as yet thrown any light upon the nature of the disease.

3. Zosteroid eruptions are not infrequently observed in cases of poisoning from coal gas, after the ingestion of arsenic, following injuries of the nerves, as a result of moral shock, as grief, or in hysteria, and probably under other conditions. These, however, are to be distinguished from the true herpes zoster as defined above.

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SOME EXPERIMENTS ON THE INTERMEDIARY CIRCULATION OF THE BILE ACIDS: A CONTRIBUTION TO OUR KNOWLEDGE OF ICTERUS.

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A LARGE proportion of the bile-acid salts that are poured into the intestine from the liver are reabsorbed. This is proved by the following four series of experiments:

1. According to different authors (Bidder and Smith, and Leyden), a dog weighing 8 kg. excretes about 4 g. of bile acids in the twenty-four hours; of this amount only 0.5 to 0.7 g. will be found in the feces (Hoppe-Seyler) and unweighable traces in the urine (Naunyn, Vogel, Höne, and Dragendorff). Leyden injected 1½ g. of one of the bile-acid salts (sodium glycocholate) into the blood of an animal and found only 0.227 g. in the twenty-four hours' urine. As the bile acids are very resistant to putrefaction in the urine and to digestive destruction in the stomach and intestine (Leyden), it cannot be assumed that they are destroyed in the bladder or the digestive tract.

2. Tappeiner placed two ligatures around a loop of intestine without disturbing its connection with its mesentery; he then placed bile-acid salts into the jejunum and the ileum at different times, and found that

the salts were completely absorbed (the loop of jejunum absorbing only the glycocholate, the ileum both the glycocholate and the taurocholate).

3. Neisser showed that nearly all the bile-acid salts that are administered by the mouth can be recovered a short time after from the bile flowing from a biliary fistula.

4. There are two principal bile acids: the one contains sulphur (taurocholic) the other does not (glycocholic). Dogs excrete the former almost exclusively, nearly 99 per cent. of the bile acids found in their bile being of the sulphur-containing variety. If, now, dogs are fed with large quantities of the sulphur-free variety it will be found that their bile (removed through a fistula) contains a large proportion of the latter, as much as from 20 to 30 per cent., showing conclusively that at least that much of the sulphur-free salt had been absorbed from the intestine to be re-excreted in the bile (Weiss). (Prevost and Binet.)

Whereas the fact, therefore, is established that the bile acids are in great part absorbed from the intestine to be re-excreted through the common duct of the liver, the path that they follow in this intermediary circulation and the function they perform *en route* are obscure.

Tappeiner succeeded in finding appreciable quantities of the bile acids in the lymph of the thoracic duct of a dog, but no one so far has found them in the blood under normal conditions. As the bile acids contained in the thoracic lymph must necessarily be poured into the systemic blood, and as they seem to disappear there only to reappear in the bile, we are confronted with the interesting problem, *What becomes of the bile acids while they are circulating from the thoracic duct to the liver?*

The following two considerations led us to assume that possibly the bile acids were present in the blood in a form that rendered them undiscoverable by known methods:

1. When isolating the bile acids from the bile it is necessary to remove the mucin by precipitation. This precipitate always contains a large proportion of the bile acids. The mucin and the bile acids are combined so firmly that it is impossible to separate the two by repeated washings or any of the other manipulations commonly employed for the separation of bile acids from organic substances; and still this combination is purely a mechanical one, for by a very long and very tedious process of dialysis it is possible to finally separate the two.

2. Bile acids are very toxic. When they are poured into the blood from the thoracic duct it is probable that they suffer the fate of other toxic substances, and are taken up by the phagocytes, which carry them through the blood-stream to the liver. Here, like many other poisons, they are liberated, arrested, and finally excreted in the bile.

These two possibilities must, therefore, be taken into consideration. Either the bile acids form compounds with some of the proteids of the serum similar to those formed with the proteids of the bile (they may

exist during life or be formed, as in the case of the bile, when the proteids are precipitated) or they enter the body of the leucocytes. In either case they would evade detection by ordinary methods.

The task set is, therefore, to isolate the bile acids from either hypothetical combination by methods other than those ordinarily employed. Here two possible manipulations suggest themselves:

1. To submit large quantities of blood to artificial digestion. This method would be based on the fact that the bile acids are more or less resistant to the action of the digestive ferments. The proteid compound would be digested away, leaving the bile acids free in the solution.

2. To oversaturate large quantities of blood with many volumes of absolute alcohol. This method would be based on the fact that the bile acids are readily soluble in alcohol, whereas all proteids (whether in or of the serum or the leucocytes) are coagulated, precipitated, and rendered insoluble by contact with alcohol.

EXPERIMENT I. Fifty c.c. of dog's blood were slightly acidulated with acetic acid and boiled. The voluminous coagulate of albumin was filtered off; the clear filtrate was examined for bile acids and found to contain none; the coagulate was divided into two equal parts, and one portion was placed into a sufficient quantity of artificial gastric juice, the other into a sufficient quantity of pancreatic juice; both mixtures were allowed to remain in the incubator at body temperature for forty-eight hours, and the flasks frequently agitated during this time. Each of the two fluids was then examined for bile acids; the result was negative.

EXPERIMENT II. Fifty c.c. of blood were allowed to flow directly from the vein of a dog into a sterile flask containing 300 c.c. of absolute alcohol warmed to 40° C. The mixture was rapidly shaken for about a minute, and the thick precipitate that formed filtered off; this coagulate was repeatedly washed with alcohol, expressed, and finally dried in the vacuum. The dry, amorphous mass was powdered and extracted with boiling water; this extract contained no bile acids. The alcoholic filtrate and the alcoholic washings were united and the bulk of the alcohol distilled off until the volume of the fluid was reduced to about 50 c.c. This fluid contained bile acids.

The last experiment shows that bile acids are present in the blood of a normal dog. Similar experiments repeated with human blood obtained from volunteers by venesection, and, in one instance, from the operating-table, showed that they are also present in human blood in normal subjects.

It was necessary to determine, however, whether the bile acids are bound to some proteid substance of the serum or whether they are carried by the leucocytes. Serum and blood-corpuscles were examined separately by the alcohol method, and it was found that the bile acids were present in the corpuscles and not in the serum. In order to determine whether the white or the red cells contained these bile acids the vein of a cow was ligated in two places, excised, and suspended for twenty-four hours in the ice-chest; a separation of the corpuscles by gravity

can be brought about in this way. If an aspirating needle is inserted into the lowest portion of this vein only red blood-corpuscles will be aspirated. These were examined by the alcohol method, but no bile acids were found. We did not succeed very well in aspirating only white cells from the higher layers, and the quantities obtained were too small to justify any conclusions; the *absence* of bile acids in the red corpuscles and the serum permits the conclusion that the bile acids are, in fact, normally carried in the leucocytes. This question cannot be definitely decided until a method is discovered of procuring large quantities of leucocytes from the blood.

If the bile acids were an excretory product pure and simple, with no further function to perform, they would not be poured into the uppermost portion of the intestine, but would probably enter the lower portion of the bowel. As a matter of fact, it is known that the bile acids perform a variety of important functions in the intestine that are fairly well understood; these do not concern us here. We are occupied with the question, *What function do they perform in the systemic blood during their intermediary circulation from the intestine back to the liver?*

The following toxic effects of the bile acids are known:

1. They have a powerful cytolytic action. Injected even in small doses they produce a wide-spread disintegration of the red blood-corpuscles, with a liberation of their hæmoglobin; brought in contact with other cells of the body they cause their disintegration.

2. They have a distinct cholagogue action—are, in fact, the only substances known to possess this power, and actually cause an increased flow of bile.

3. They aid coagulation in small doses (1 : 500).

4. They stop coagulation in large doses (1 : 250 and over).

5. They slow the heart-beat by a direct action on the heart muscle and the cardiac ganglia.

6. They act as vasodilators in very small doses.

7. They act as vasoconstrictors in large doses.

8. They reduce motor and sensory irritability.

9. They act on the higher cerebral centres, causing coma, stupor, and death from "gentle enslumbering."

Of all these possible effects only those can normally take place in the body that can be exercised in doses as small as those normally found in the blood, namely, those described under 1, 2, 3, and 6.

We are justified, therefore, in assuming that, aside from their intra-intestinal functions, the bile acids are concerned in the normal destruction of cells, chiefly red blood-corpuscles; that they exercise an influence on the flow of bile; that they aid coagulation and play a rôle in vasodilatation; in pathological states, when large quantities of the bile acids enter the blood, their other powers are developed and become clinically manifest.

The finer mechanism of these various activities has so far not been made the subject of experimentation.

CLINICAL EPICRITICISM. The discovery of the bile acids in normal blood has a definite and important bearing on the origin of icterus.

Notwithstanding the fact that under certain pathological conditions (hemorrhagic exudates and extravasations, subcutaneous injections of red blood-corpuscles or hæmoglobin, pyæmia without occlusion of the bile-ducts or other impediment to the flow of bile), bile-pigment is found in the tissues, enters the blood, stains the tissues diffusely, and is excreted in the urine, the existence of extrahepatic "hæmatogenous," or, better, "anhepatogenous" icterus is almost universally denied. It is claimed that every icterus is an icterus from absorption of bile from the liver; that blood-pigment is converted into bile-pigment in the liver alone, and that even in excessive hæmolysis or after injection of hæmoglobin, icterus (hæmato-hepatogenous) is due to the increased viscidity of the bile causing occlusion of bile-channels, stasis, and absorption, or to some injury of the hepatic cells that permits diapedesis of bile-constituents from the bile-channels through the liver cells to the blood-channels.

The chief argument adduced in favor of this view is the presence under these conditions of bile acids in the blood and urine, for, it is claimed, these are a specific liver product formed only in the liver and by the liver, and *never normally present in the blood*. Aside from the fact that a series of experiments that I have recently published show that bile acids are not a specific product of the liver cells, and make it highly probable that they can be and are normally, and to a greater degree pathologically, formed outside of the liver, the investigations reported above show that they are normally present in the blood; so that this argument and the conclusions that are based on it collapses.

If, *per contra*, the adherents of the theory of hæmatogenous icterus claim that every icterus in which bile acids are absent from the blood and urine is hæmatogenous ("Finden sich keine Gallensäuren—dann haben wir hematogenen Icterus vor uns," Leyden) they, too, occupy an extreme position, for we know that in some cases of the most pronounced obstructive icterus in which there is occlusion of ducts, stasis of bile, absorption of bile constituents, etc., ordinary methods may fail to reveal the presence of bile acids in the blood and urine. *They are, however, never absent from the blood.*

All arguments, therefore, that are based on their "presence" or "absence" from the blood are essentially fictitious and invalid.

From the bile-acid findings in any given case we are no longer justified in categorically asserting or denying the existence of pure hæmatogenous icterus, nor are we aided by these findings in formulating any definite diagnostic conclusions in regard to the participation of the liver or its ducts in any disease-process complicated by icterus.

REVIEWS.

A MANUAL OF SURGICAL TREATMENT. By W. WATSON CHEYNE, C.B., M.B., F.R.C.S., F.R.S., Professor of Surgery in King's College, London, and F. F. BURGHARD, M.D., M.S. (Lond.), F.R.C.S., Teacher of Practical Surgery in King's College, London. In seven volumes. Vo'ls. I. to V. Philadelphia and New York: Lea Brothers & Co.

THE original intention was to issue this book in six volumes, but the subject grew in the writing, and it became necessary to obtain enlarged space. The fifth volume of this valuable series is now before us, and is equal in merit to its predecessors. It deals with the head, face, jaws, lips, larynx, and trachea, and the intrinsic diseases of the nose, ear, and larynx, the latter subjects being discussed by H. Lambert Lack, M.D.

An extensive work upon surgical treatment was greatly needed, and the manual by Cheyne and Burghard fills a long-realized want. In the five volumes so far issued the authors have considered thoroughly, carefully, and sometimes even elaborately, the treatment of injuries and of surgical diseases. The constant effort has been to furnish clear descriptions and detailed information, and the result is singularly gratifying. Often when dealing with a difficult case—a case in which ordinary methods of treatment have proved futile—have we wished for a book like this, because the information upon treatment contained in the ordinary text-book is usually scanty and incomplete, and is sometimes so meagre as to be practically useless. The authors of this manual assume that the reader is familiar with the nature and diagnosis of the disease, and "refer to pathology and symptoms" only so far as is necessary to render "intelligible the principles on which treatment is based and the various stages of the disease to which each particular method is applicable." They do not set forth all methods or even many methods of treating each condition, but prefer to describe the treatment which has been most successful in their hands.

In treating acute inflammation the authors do not particularly advocate general blood-letting except in diseases of the lungs and brain with enlargement of the right heart. They use cold only early in an inflammation, but fail to caution us against the intermittent use of cold. They point out dangers in the employment of Leiter's tubes, emphasize the fact that heat is used to treat inflammation after cold has been abandoned, and that heat is not applicable to a skin inflammation. Chronic Inflammation is discussed in a separate chapter, and in this article the value of free incision is especially dwelt upon.

In the section upon Acute Abscess, Hilton's method is advocated for reaching deep-seated collections of pus in certain regions, and we are told that when Hilton's method is used a probe must be inserted before the forceps are withdrawn, so that the opening in the deep fascia will

not be lost. Particularly judicious is the direction to avoid violent squeezing of an abscess after opening, because squeezing may injure the abscess wall and lead to dissemination of the infection. Washing out an acute abscess after incising it is strongly condemned on the grounds that irrigation cannot satisfactorily disinfect, and may injure the abscess wall, lead to diffusion of the infection or the production of a weak spot in which micro organisms "which would otherwise die can spread."

In the section on Ulcers, skin grafting by the methods of Reverdin and Thiersch is carefully described, but the transplantation of pieces of the entire thickness of the skin is not discussed.

We are advised to cover the grafted area with strips of protective, one inch wide (the strips having been sterilized in 1 : 20 carbolic-acid solution and washed in boric-acid solution), and to apply over the protective, first, cyanide gauze wrung out of a solution of corrosive sublimate (1 : 4000), and then salicylic wool. In from five to seven days the dressing should be removed and a fresh dressing applied. At the end of another week dressing can be dispensed with.

In senile gangrene of the foot, Cheyne and Burghard believe that usually amputation through the knee-joint is the proper procedure.

In diabetic gangrene they advocate early amputation high up, and they do not believe that coma is more apt to arise if amputation is performed than if the gangrene is allowed to go on undisturbed.

The article upon Anæsthetics is most excellent and practical. The author is J. Fred. Silk. He doubts the value of morphine given before the administration of an anæsthetic, and believes the drug may be actually dangerous by masking the symptoms of overnarcosis.

In Silk's article the Subsection upon Special Cases is of decided practical use.

The article upon Local Anæsthesia is short and unsatisfactory, and does not do justice to a most valuable expedient.

In the section dealing with wounds the authors point out that fibrin ferment is a useful agent to check capillary oozing. They advocate the use, in certain regions, in order to avoid scars, of buried interrupted sutures passing through the derm but not the epiderm. Such sutures seem to us clumsy and troublesome of application when compared to Halsted's admirable subcuticular stitch.

In the article upon Nævi the clearest description we know of is given of the application of electrolysis.

In treating of aneurisms of large vessels we are told that the stay knot is reliable and satisfactory. The authors have evidently little faith in wiring aortic aneurisms, and prefer distal ligation to Macewen's method of needling.

The sections on the Skull and Brain are notably good, and we are glad to find that this book does not advocate the treatment of microcephalic idiocy by the ridiculous operation of linear craniotomy.

In cleft palate the authors adopt the modern view of Owen, that comparatively early operation is advisable. They do not delay operation after the third year.

In the section on Fractures no attempt at complete enumeration of methods is attempted, as the authors only speak of those which have proved useful in their hands. In this section it becomes evident that exploratory operations and operative fixation of fragments are coming

more and more into vogue. The operative treatment of fracture of the patella is warmly advocated.

The above is a very hasty and imperfect sketch of some of the views set forth in this useful and scientific book.

The *Manual of Surgical Treatment*, although it omits some valuable methods, is, in our opinion, one of the most practically useful of recent publications. We employ it frequently and warmly commend it. The scope of it will be best realized by noting the subjects treated in each volume.

Vol. I.: The treatment of general surgical diseases, including inflammation, suppuration, ulceration, gangrene, wounds and their complications, infective diseases and tumors; also an article on the administration of anæsthetics, by Dr. Silk.

Vol. II.: Treatment of the surgical affections of the tissues, including the skin and subcutaneous tissues, the nails, the lymphatic vessels and glands, the fasciæ, bursæ, muscles, tendons and tendon sheaths, nerves, veins, arteries, and deformities.

Vol. III.: Treatment of the surgical affections of the bones, and amputations.

Vol. IV.: Treatment of the surgical affections of the joints (including excisions) and the spine.

Vol. V.: Treatment of the surgical affections of the head, face, jaws, lips, larynx and trachea, and the intrinsic diseases of the nose, ear, and larynx.

These five volumes contain over 1800 pages and nearly 600 illustrations.

J. C. Da C.

Practical Surgery: A Work for the General Practitioner. By Nicholas Senn, M.D., Ph.D., LL.D., Professor of Surgery, Rush Medical College, Chicago. Handsome octavo volume of 1133 pages, with 650 illustrations, many in colors. Philadelphia and London: W. B. Saunders & Co., 1901.

This is Dr. Senn's latest and, in some respects, most elaborate and pretentious work. It does not pretend to cover the whole field of surgery, but is supposed to contain much or most of that which is most important and most practical. In the preface the announcement is made that its contents are devoted to those sections of surgery which are of especial interest to the general practitioner. Inasmuch, however, as a large part of the volume is given up to a discussion of topics such as gunshot wounds and others quite within the domain of military surgery, we do not see exactly how this claim has been substantiated. To be sure, injuries and acute surgical diseases do usually come first under the notice of the general practitioner, but this is not true of gunshot wounds.

The volume opens with a chapter on Emergency and Military Surgery. The chapter is, in large measure, a homily in which are dealt with the qualifications and duties of the military surgeon, with remarks upon military spirit, courage, personal habits, etc., all of which make interesting reading, although their insertion here is a matter of questionable taste.

In the chapter on Traumatic Shock the subject is exceedingly well handled, both from its theoretical and practical aspects.

The following chapter is on General Anæsthesia, in which are fully discussed the accidents which may happen during narcosis, the methods of their treatment, the merits of various anæsthetics, etc. Under the head of local anæsthesia, however, there is no mention of intraspinal cocaine injection, and lumbar puncture is but briefly alluded to much later in the volume. Surely the method deserves description in such a work as this, even though the author may be averse to its use. There is nothing in the book, however, which would indicate what his views are in regard to this subject of great recent interest. Neither is there any mention made of nitrous oxide gas, nor of its use in combination with oxygen, or as a preliminary to the administration of ether, nor is the combination of chloroform with oxygen gas mentioned. In this respect there is an important omission which should be remedied in the next edition. The subject of prophylactic hæmostasis is carefully considered, including the disagreeable consequences which may follow the employment of too tight constriction, such as paralysis, etc. Angiotripsy receives very scant mention. The treatment of hemorrhage leads abruptly into a discussion of sterilization of ligature materials, and then to the practical application of ligatures to the principal vessels, to illustrate which a few of Zuckerkandl's colored plates have been introduced.

The sixth chapter is devoted to the subject of Wounds and Wound Infection, which leads the author without break into the consideration of the proper operating-room, and then of hand disinfection, etc. While insisting on the necessity of careful hand disinfection, there is no allusion to perhaps the most admirable means for accomplishing this in the use of Schleich's marble-dust soap, with its most efficient qualities. Again, in the list of various antiseptic materials which may be used we find no allusion to the silver salts, which are now coming into such general use. Neither is intravenous injection of Crédé's soluble silver mentioned in the treatment of septicæmia.

Abruptly following the chapter on gunshot wounds, and sandwiched in between it and that on fractures, is a chapter on Rupture of the Urethra, with which no fault can be found except that in this particular location it seems very much out of place.

Then follow about 250 pages devoted to the general subject of fractures, which is dealt with in the most thorough manner, and which will constitute most interesting reading for everyone, surgeon or general practitioner. Almost all possible contingencies have been taken up and dealt with, and these pages bristle with practical hints of great value. It must be said that some of the methods of treatment described are now obsolete and of historical interest only, and yet the general practitioner is tempted always to exclude the historical side of these subjects and to think only of what can be done now and not of what was formerly done for the relief of such surgical lesions. Delayed union and false joint are fully considered, and there is displayed throughout a very wide range of knowledge of and familiarity with the subject in all its aspects. We are glad to see justice done to the views of Moore, of Rochester, N. Y., whose researches on fractures and dislocations are not widely enough recognized, and who is a pioneer in many of these investigations. Skiagrams are very well used where they will prove of service, and add great value to the text. Fractures of the skull are not

separated from other fractures, as is usual in discussing regional surgery, but are brought in with the various inseparable considerations concerning intracranial injuries, which, however, are only incidentally discussed. Particularly good is the discussion on bone suture and the union of fragments in compound and comminuted fractures. The writer is not familiar with any other text-book in which so good an idea can be obtained of this important matter.

Chapter XII. deals with Dislocations, and is very complete so far as it goes. The author, however, apologizes for what seems a very great inconsistency in that dislocations of the shoulder-joint are discussed at great length and with great care, and yet no allusion is made to dislocations of the hip-joint. In spite of Dr. Senn's statement that he has seen so many fractures of the neck of the femur mistaken for dislocation that he felt called upon to emphasize description of this particular kind of dislocation, this can hardly be considered a sufficient reason for omitting all allusion to the hip dislocations, which are almost equally important and often more difficult of management. It would seem as if lack of time or haste in preparation were the real cause for this omission rather than other considerations.

Exploratory puncture, subcutaneous medication, paracentesis, drainage of joints, and aseptic catheterization follow the chapter on Dislocation, without any particular logical order, although the pages are full of practical discussion and direction; then come emergency operations on the air-passages and description of empyema and its treatment. The author is not quite correct when he says that empyema is a term used to designate the presence of pus in the pleural cavity. The term properly means the presence of pus in any pre-existing cavity, and he should have added that, by common consent, when no particular cavity is specified the pleural cavity is understood. He defines it as representing a pathological product of either a primary or a secondary suppurative pleurisy. In discussing the causes of suppuration he introduces a number of interesting facts calculated to maintain his views that the inhalation of dust is a very important causative factor, the dust acting either as a mechanical irritant or because of its contained microbes. He gives an admirable description of Schede's plastic operation upon the chest, but says very little about Estlander's less formidable and often equally serviceable method.

Chapter XVII. is a discussion of the general subject of Peritonitis, and constitutes one of the most valuable chapters in the book. He classifies the disease anatomically, then etiologically, then pathologically, then bacteriologically, and, finally, clinically. It probably is the most elaborate treatment which the subject has received in any text-book. He then proceeds to discuss the handling of each of the clinical forms, which naturally leads him into the discussion of eventration, irrigation, incision of distended bowel, abdominal drainage, and after-treatment; incidentally, also, but fortunately, the subjects of perforating ulcer of the stomach and duodenum, as well as of the typhoid lesion of the small intestine, are dealt with, and the importance of their operative treatment not minimized. It is interesting to note that Senn discusses a so-called hæmatogenous peritonitis, which he describes as occurring without injury or discoverable lesion, and consequently as the result of infection through the blood. It has been observed most often in connection with nephritis, pyæmia, rheumatic arthritis, and the acute

exanthems. A careful perusal of these pages on the general topic of peritonitis will amply repay the time spent. Appendicitis is discussed in the same complete and thorough manner as peritonitis. Its various forms are fully described, and, indeed, well illustrated with a colored plate. The author believes that its rational treatment should depend entirely on its anatomico-pathologic form. Its drug treatment is discussed only from the surgical point of view, which is probably the correct position to take. Intestinal obstruction is the subject of an important chapter which is devoted, however, rather to the therapeutic aspect than to the anatomical or pathological aspect of the topic. The various operations are well described.

A general chapter on Abdominal Section is made to include a large number of the intra-abdominal operations, intestinal suture, anastomosis, etc., while a further special chapter is given to a particular consideration of the latter suture alone. All forms of anastomosis and implantation are in this chapter discussed and suitably described. The next chapter takes up the various pathological forms of Intestinal Obstruction, which are here treated at considerable length and suitably illustrated by diagrams and plates. The various impactions by foreign bodies and their suitable treatment are also described. Intraperitoneal tuberculosis, aside from that of the peritoneum, here finds consideration extending over some thirty pages, which are all replete with interesting statements. Intestinal obstruction after abdominal section is also considered. To the subject of strangulated hernia some twenty pages are devoted, while intestinal fistula receives about the same number, the various pathological causes for the same being fully rehearsed. The concluding portion of the volume, some eighty pages, is devoted to exsection of joints and amputation of limbs. It cannot be said that these subjects are completely dealt with by any means, but rather treated by general considerations. To what has been said no exception can be taken. One is tempted only to wish, as he reads, that more complete discussion had been given them.

Altogether the book can be regarded as representing the results of many years of most extensive and varied surgical experience. As such it is an extremely valuable contribution. No one can study it without realizing its value, and, at the same time, without wishing that the author might have been more explicit or extended in certain respects. Such a regret must always be voiced when such an author deals but partially with a given subject or but incompletely with the entire list of surgical topics. A regret thus expressed is, however, the highest compliment that can be given to a work. Altogether, the reviewer regards the book as the best product of Dr. Senn's brain and activity, and bespeaks for it wide-spread attention. R. P.

OPERATIVE SURGERY. By JOSEPH D. BRYANT, M.D., Professor of the Principles and Practice of Surgery in the University and Bellevue Hospital Medical College, etc. Vol. II., containing 827 illustrations, of which 40 are colored. New York: D. Appleton & Co., 1901.

THE second volume of Dr. Bryant's work on *Operative Surgery* seems to us even more full and commendable than the first. He begins with operations on the mouth and respiratory passages. There has been no

omission of any of the operations on the tongue or mouth which can in any way detract from the value of such an encyclopædic work. The various methods of pharyngotomy are succinctly and well given. Operations on the nose are more fully described than usually, save, perhaps, some of those plastic operations for minor deformity and for purely cosmetic results, which might find more extensive mention. Operative surgery of the œsophagus is fully dealt with, including all the newer operations for stricture, most of which are well illustrated. Tubage of the œsophagus also is described more fully than in almost any other work with which we are familiar. Its complete description, however, does not commend it to the practising surgeon, and the statement that of seventeen cases treated by the method nine died, without obstruction, from the effects of the advance of the disease, is scarcely enough to seriously commend it.

The chapter on Operations on Viscera Connected with the Peritoneum is admirable. It is opened by general considerations, including the toilet of the peritoneum, intra-abdominal drainage, after-treatment, etc. The author then proceeds at once to operations on the intestine, in which are described not quite all the various sutures that have ever been devised, but all that have proven of sufficient value to commend them to the surgeon, and more, in fact, than the ordinary operator can well bear in mind. Intestinal anastomosis by various methods is shown, and the various instruments which have been recently invented for this are sufficiently described. A most valuable addition to the ordinary minute directions for operations will be found in certain sections like that devoted, for instance, to the general operative treatment of gunshot and contused wounds of the abdomen. These are cases which demand all the judgment of an experienced operator, and such judgment has been evinced in general treatment of the subject by Dr. Bryant's discussion. Intussusception, along with other forms of intestinal obstruction, is fully dealt with, both in the text and pictorially.

No such work as this could neglect the general subject of appendicitis, to which a number of pages are devoted, with admirable directions for practitioners, under varying conditions, both acute and chronic. A full description of the various modified incisions and discussion of their advantages are given. The surgical treatment of peritonitis and of perforating ulcer is also fully described. The section on operations on the stomach is preceded by a colored diagram, with discussion of certain anatomical points which one must have ever in mind in order to become a successful operator upon this viscus. Numerous methods of gastrostomy are illustrated, and the author then proceeds to the various ways of bringing about anastomosis between the stomach and bowels. These pages are amply illustrated, and the directions are made so plain that no one can misunderstand them. Surgery of the liver, especially of the gall-bladder and biliary passages, is admirably treated, and the illustrations in this regard leave nothing to be desired. The same may be said of operations upon the kidneys. Modern surgery of the ureters is not neglected, and the reader will find here an epitome of practically everything that has been done in this direction. Hernia is fully treated and numerous methods described. Here, as in some other instances, more methods are given than any one surgeon can well familiarize himself with, and certainly more than anyone ought to practise. Nevertheless, enough is said of all in order to allow an intelligent

reader to select that of which he will most often avail himself. We are particularly struck by the wealth of description of methods of treatment for prolapse of the rectum, everything of known value being mentioned, and even fully described. The various methods of attacking cancer of the rectum are given sufficient space, although the author is not quite so full in this regard as he has been in most others. Operations on the breast are fully described, and the proper plea for thoroughness of work is not omitted.

Surgery of the lung is given its due proportion of space, and that nothing is neglected one may see when even the resection of a tubercular deposit in the lung is described. The author's method of attacking the mediastinum from the dorsal region is furnished, and is made to appear in its full value. The chapter upon Operations on the Neck is full of value and sufficiently illustrated. The chapter upon Operations on the Genito-urinary Tract is exceedingly well illustrated, and we scarcely discover the omission of any operative feature which has any merit whatever. Among the operations on the scrotum and penis one might, perhaps, look in vain for mention of Beck's recent work, and yet this has been, perhaps, too recently published to find mention in this volume. The book closes with miscellaneous operations, among which especially are to be mentioned those upon the patella, which are well described, and the various methods of uniting broken bones by operative or mechanical intervention. Excision of the cervical sympathetic is also included here, instead of under operations on the neck, the description being complete and admirable, and the results summarized sufficiently to enable one to judge of its merits.

Altogether, the work is most admirable and indispensable to every operating surgeon of large practice. As the reviewer compares it, in his mind, with other works covering the same ground, he cannot think of one to which he would wish to turn so often as to this monumental work of Dr. Bryant's. R. P.

ESSENTIALS OF REFRACTION AND OF DISEASES OF THE EYE. By EDWARD JACKSON, A.M., M.D, Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. Third edition, revised and enlarged. 12 mo., 261 pages, 82 illustrations. Philadelphia and London: W. B. Saunders & Co., 1901.

THIS, the third edition of Jackson's well-known work, the best of its kind, has undergone most careful revision and extension; much that is new has been added, while the entire subject-matter has been brought to date.

For both the ophthalmic student and the busy practitioner who wish to obtain a succinct general account of diseases of the eye, the book can be heartily recommended. C. A. O.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Case of Family Periodic Paralysis, with a Critical Digest of the Literature.—SINGER and GOODBODY (*Brain*, Summer, 1901, p. 257) report a case of periodic paralysis in a boy, aged sixteen years, who was admitted to the National Hospital, London, on October 12, 1900. There was no history of any similar trouble nor of any neuropathic tendency in his family. The boy's first attack occurred when he was fourteen years of age, and the second four to six weeks later. The attacks increased in frequency until they occurred almost weekly, when the boy first came under observation. They had practically always come on at night. The writers describe one of the attacks in detail. It began at 3 P.M., with a dull, aching sensation and weakness in the legs. One hour later weakness of the upper extremities began. By 7.30 the muscles of the neck, trunk, and extremities were almost completely paralyzed. The respirations were almost entirely diaphragmatic. The facial muscles and cranial nerves were not affected. There was no mental disturbance. The deep reflexes disappeared *pari passu* with the loss of power. The abdominal and cremasteric reflexes were absent, and the plantar diminished. During the night muscular power gradually returned, and by 10.30 A.M. the following morning he was practically well. The order of recovery was the reverse of the order of involvement—that is, the last muscles to lose power were the first to regain it. Electrical examinations in this and subsequent attacks showed that during an attack there is a gradual reduction of the excitability both to faradism and galvanism until, with complete paralysis, there was complete loss of excitability to both currents. The heart in this case was often dilated during the attacks.

The urine and feces were carefully examined. The toxicity of the former, as tested on rabbits by Bouchard's method, was much increased during the attack, but no attempt was made to isolate the toxin. The experiments on the feces were negative.

Sections of the left sartorius muscle excised when it had completely lost its power to contract showed an extraordinary tendency to fissuring of the individual muscle fibres. This was regarded as an artefact, but was held by the writers to indicate that the muscle had undergone changes, owing to the fact that normal muscle did not give the same picture when the same technique was followed.

The first case of the disease was reported by Hartwig in 1874. The affection is comparatively rare. There is usually a marked family history. This is illustrated by the cases recorded by Cousot, Goldflam, Taylor, and Mitchell, who give the number of relatives affected as five, nineteen, eleven, and seven, respectively. Goldflam and Taylor have both traced the disease back through five generations. The age of the patients at the onset of the first attack has ranged between six and twenty-four years. Both sexes are equally affected The attacks have almost invariably begun at night. The intervals between the attacks gradually diminish until they occur every week or even oftener. The weakness nearly always begins in the legs, extending to the arms, and lastly to the trunk and neck. During the height of the paralysis the intercostals and extraordinary muscles of inspiration are paralyzed. The diaphragm escapes. The heart is often dilated, at times irregular in action, and occasionally a systolic murmur is heard over it. There is loss of excitability of the muscles to faradism and galvanism during an attack, but normal electrical excitability in the intervals.

Regarding the etiology, there is a general consensus of opinion that the affection is due to the action of a toxin. Whether this is a specific toxin or an increase in some of the normal products of metabolism has not been definitely determined. Life does not seem to be shortened by the disease, and death does not appear to have resulted from the attacks. The number of the attacks diminishes toward middle age, and sometimes they cease altogether.

The treatment has hitherto proved unsatisfactory. The writers recommend that the patient should live a quiet, non-exciting life, with plain, simple food and moderate physical exercise. Diuresis should be promoted by large quantities of water and the administration of digitalis and potassium acetate or citrate.

Babinski's Toe Phenomenon.—SCHNEIDER (*Berlin. klin. Wochenschrift*, 1901, xxxviii., 946, as a result of observations in a variety of different conditions in the Clinic of Professor Jolly, comes to the following conclusions:

The normal reflex movements on irritation of the sole of the foot consist of two reflexes which arise from different points in the central nervous system. The first, simple plantar flexion following a weak stimulus, is a cortical reflex. The second, dorsal flexion of the toes with combined movement of the leg on strong stimulus, is a spinal reflex.

Babinski's phenomenon consists in the fact that, upon a feeble stimulus, plantar flexion does not follow, while dorsal flexion of the toes—that is, a spinal reflex—immediately appears. That this is more easily obtained than under normal circumstances depends always upon a general increase of the spinal reflexes.

Babinski's phenomenon may arise in two ways: First, through interrup-

tion of the pyramidal tract, and consequent obliteration of the cortical reflex; this interruption may also occur at the beginning of the tract—that is, the motor cortex (epilepsy). This reflex, depending upon organic changes, the author describes as "genuine."

Secondly, if, as a result of general increase of the spinal reflexes (strychnine, diseases resulting in increased reflexes without lesions of the pyramids), or from conditions which diminish the reflex irritability of the cortex (stupor, œdema), the spinal reflex becomes as easily or more easily obtainable than the cortical reflex, then as a result of the dynamic excess of the spinal reflex the cortical reflex may be suppressed and apparently wholly wanting. Thus the typical picture of Babinski's reflex may arise without lesion of the pyramidal tract.

We are not in a position to separate positively this variety of Babinski's phenomenon, with suppression of the cortical reflex, from that with destruction of the cortical reflex; it is then impossible, in a condition of general increased reflex irritability, to make the diagnosis of a pyramidal lesion from Babinski's phenomenon alone. However, permanent persistence of Babinski's phenomenon is rare, without destruction of the cortical reflex (a single plantar flexion among a large number of dorsal flexions shuts out a genuine Babinski reflex); and since the genuine Babinski's phenomenon is present or develops almost always in lesions of the pyramidal tract, this reflex is of practical value in separating pyramidal lesions from similar pictures.

The Alkalinity of the Blood in Physiological and Pathological Conditions.—Orlovsky (*Vratch*, 1901, xxii., 1190, 1222), after a review of the literature and control tests of various methods, gives the results of a considerable number of observations on animals and human beings which lead him to the conclusion that:

1. The previous methods of determining the alkalinity of the blood give varying results even in healthy individuals, and should, therefore, be finally abandoned.

2. Constant results are only to be obtained by methods which determine the alkalinity in the laky blood; these methods alone have a *raison d'être*.

3. Variations in the alkalinity of the blood noted in various diseases and in one and the same disease by various investigators working according to old methods (Landois-Jaksch's, etc.), depend upon the varying isotonicity of the blood and its varying richness in red corpuscles in different cases.

4. The variation of the alkalinity of the blood noted by authors employing titration of laky blood depends upon the variation in number of red blood-corpuscles.

5. The alkalinity of the blood-plasma is markedly diminished in cancerous cachexia, in advanced uræmia, and in severe diabetes mellitus.

6. In other diseases the alkalinity of the blood-plasma remains unchanged, or, if it does differ from the normal, shows but slight and exceptional variations.

The diminution in the alkalinity of the blood in uræmia, diabetes, and cancerous cachexia depends upon the accumulation in the blood of acid products of metabolism. The direct dependence of diabetic coma upon an

acid auto-intoxication is generally recognized, and the use of alkalies internally is, as is well known, often advised. The author has carried on a series of experiments upon the normal subject to determine the best method of administering alkalies. As a result of investigations in six cases, he finds that: (1) a single dose of bicarbonate of soda produces a relatively unimportant and very transient increase in the alkalinity of the blood; (2) warm alkaline enemata increase the alkalinity of the blood appreciably more than the administration of corresponding quantities of alkalies by mouth; (3) this increase in the alkalinity of the blood is slight and of short duration.

In two cases of diabetes the author was able to show clearly that: (1) the administration of alkalies increases the alkalinity of the blood and of its plasma to an appreciably higher degree than in healthy individuals; (2) warm alkaline enemata increase the alkalinity of the blood and of its plasma appreciably more than doses of alkalies of the same quantity by mouth; (3) this increase in the alkalinity of the blood is of short duration, but lasts longer when the alkalies are administered by rectum.

The author believes that these results are important as indicating the advisability of treatment by alkaline enemata at the first evidence of diabetic coma. The administration of alkali should be continued well after the removal of the immediate danger, as otherwise the alkalinity of the blood will rapidly fall again.

On the Influence of Formic Aldehyde upon the Metabolism of Children.—TUNNICLIFFE and ROSENHEIM (*Journal of Hygiene*, July, 1901, p. 321), as a result of very careful experiments on the effect of formic aldehyde on the metabolism in children, as well as from a review of the literature, come to the following conclusions:

1. In healthy children formic aldehyde administered with the food in doses up to 1 : 5000 in milk, or 1 : 9000 in total food and drink, exerted no appreciable effect on the nitrogen or phosphorus metabolism or fat assimilation. The analytical figures suggest, however, that formic aldehyde has a tendency to diminish phosphorus and fat assimilation, and hence it may be inferred that in larger doses, or if continued for a longer period, it would act in this direction. This effect is referable to an influence upon pancreatic digestion.

2. In healthy children formic aldehyde in the above doses produces a retention of water in the body.

3. In a delicate child formic aldehyde in the above maximum dose had a chemically measurable deleterious effect upon the nitrogen, phosphorus, and fat assimilation, again referable to an action upon the pancreatic digestion, combined with a slight intestinal irritant action. There was a slight tendency to stimulate the katabolism of proteid material.

4. In a delicate child formic aldehyde increased the volume of urine and the weight of feces.

5. In all cases the excretion of lecithin in the feces was diminished under the influence of formic aldehyde. This effect is probably referable to a stimulating action of formic aldehyde on the lecithin-splitting ferment of the pancreas.

6. In no instance did formic aldehyde exert any appreciable intestinal antiseptic action.

7. In no instance was there any influence on the general health or well-being of the children.

The Intrapleural Pressure in Pneumothorax.—BARD (*Revue de Médecine*, 1901, xxi., 441, 576), in an interesting communication which well repays careful study, reaches conclusions which are best stated in his own words:

1. Owing to the anatomical structure of the lung, wounds of its parenchyma of most varied and extensive nature are permeable to gas and liquids only in the direction from the bronchi to the pleura. Only grave and extensive lesions of the organ or absolute obstacles to the retraction of the tissues, such as are found in partial pneumothorax, may permit the passage of gas or liquids in a direction from the pleura to the trachea.

2. In generalized pneumothorax, when the fistula is not obliterated, the pressure of the gases is positive during both periods of the respiration in quiet breathing. This positive pressure results from the spontaneous tendency toward the equilibrium of the intrabronchial and intrapleural gases by the regular play of respiration, without any part being played by the cough. The hypothetical pneumothorax with a valve is the normal form of general pneumothorax open internally.

Cough and efforts act on the intrapleural pressure more energetically than on the intrabronchial pressure. In this manner they contribute toward compressing the lung and rendering it atelectatic, toward pushing over the mediastinum, but in no way toward allowing the penetration of air into the pleura through the pulmonary wound.

The degree of positive intrapleural pressure is but slight and practically constant in a given patient, presenting respiratory oscillations of several centimetres on either side of a mean of about 6–8 cm. of water. After accidental perturbations it tends gradually to re-establish itself at its fixed level by the normal play of the respiration.

3. The degree of this pressure is, if not equal to the pulmonary elasticity, at least in relation with it and determined by it. This elasticity, which, combining with the action of the walls, brings about a negative pressure in the pleural cavity when the lung is intact, provokes, on the other hand, in pneumothorax with persistent pulmonary opening, a positive pressure, owing to its direct transmission along the fistula.

4. The positive intrapleural pressure in pneumothorax is a phenomenon of pathological adaptation and compensation in relation to the persistence of the fistula, in that it assists in obliterating it; it should be respected when present, and one might well hasten its production during the phase of initial suffocation.

5. Mensuration of the pressure of the intrapleural gases is an element essential for the diagnosis of the varieties of pneumothorax, and principally for the appreciation of the existence of a pulmonary fistula, its persistence, or its obliteration. However, the character of the mean pressure, whether positive, nil, or negative, does not furnish exact indications for diagnosis; one should substitute for this the consideration of the extremes of pressure. The pressure is positive at both periods in generalized pneumothorax with

persistent fistula; it is positive on expiration and negative on inspiration in partial pneumothorax with open fistula; it is negative in both periods in general pneumothorax when there is no fistula or when this has been obliterated for a greater or less length of time.

SURGERY.

UNDER THE CHARGE OF

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Pancreatitis.—MAYO ROBSON (*British Medical Journal*, May 11, 1901) states that the essential and immediate cause of the various forms of pancreatitis is bacterial infection, this having been positively proved both clinically in the human subject and experimentally in the lower animals. External determining causes are biliary and pancreatic lithiasis, injury, gastro-duodenal catarrh, ulcer and cancer of the stomach, pylorus or duodenum, and zymotic diseases, such as typhoid fever and influenza, and, in some cases, pancreatitis has come on suddenly in persons of robust health, and the determining cause has been beyond recognition. Fat necrosis is commonly found in association with pancreatitis and other diseases of the pancreas, but it is not pathognomonic of disease of the pancreas. Hemorrhage into the pancreas not due to injury may occur, and there is an ill-understood relation between pancreatic disease and serious hemorrhage. Careful investigation has shown (1) that in certain diseases of the pancreas there is a general hemorrhagic tendency which is much intensified by the presence of jaundice; (2) that hemorrhage may apparently occur in the pancreas unassociated with inflammation or with jaundice, or with a general hemorrhagic tendency; (3) that both acute and chronic pancreatitis can and do frequently occur without hemorrhage; (4) that some cases of pancreatitis are associated with local hemorrhage. The treatment of acute infective pancreatitis is practically that of peritonitis, commencing in the superior abdominal region. The pain at the onset is so acute as to necessitate the administration of morphine for its relief, and collapse will demand stimulants, which, on account of the associated vomiting, may have to be given by enema. In the early stages the symptoms are usually so indefinite as not to warrant operation, and until the collapse has passed off no surgical procedure would generally be justifiable. The simulation of intestinal obstruction will probably lead to efforts to secure a bowel movement, and so relieve the distention. An early evacuation of the septic matter is necessary to recovery, so an exploratory operation is demanded so as to evacuate the septic material and give free drainage. The after-treatment should be

directed to combating shock and keeping up the patient's strength. Even if pus is not found, the exploratory operation will do no harm. The incision is best made in the median line above the umbilicus, and should enable one to establish the diagnosis. The best incision for drainage is a free vertical incision in the left costo-vertebral angle. There is then no risk to the general peritoneal cavity, and drainage will be good, because it will be dependent. Subacute pancreatitis is more amenable to treatment—morphine for the pain, calomel as an intestinal antiseptic, and for the relief of the distention. As soon as the constipation is relieved, diarrhœa is apt to supervene, and this should have its appropriate treatment. If surgical treatment be decided upon either the median incision above the umbilicus or the posterior incision in the left or right costo-vertebral angle should be employed. If a definite abscess forms and approaches the surface in front of or in either loin, the treatment will be incision and drainage, as in the case of any other abdominal abscess. The author reports five cases of subacute pancreatitis treated by operation, with the result of three recoveries and two deaths. Chronic pancreatitis must be treated by abdominal section and drainage, but in these cases the drainage should be indirect, and obtained by draining the gall-bladder by cholecystotomy, cholecystenterostomy, or duodeno-choledochotomy. The exact line of treatment cannot be determined until the abdomen is opened. When there are gallstones present they should be removed, unless the patient is too ill to permit of the complete operation; but in every case drainage must be secured, if possible, by cholecystotomy; and, moreover, the drainage must not be stopped before the bile has become healthy, and not before the greater amount of bile is being passed by the bowel, which will be certain to occur as soon as the swollen pancreas has subsided, if the duct be otherwise clear of obstruction. The simulation of malignant disease of the head of the pancreas by chronic interstitial pancreatitis would make the author hesitate to decline operation in any case of distended gall-bladder where the patient is in a condition to bear it, or even in any case of chronic jaundice without distention of the gall-bladder where the general health is deteriorating, as though it should be recognized that if the disease be really malignant very little good will be done, and life may even be shortened or only prolonged for a short time, yet if the disease prove to be chronic pancreatitis a real and permanent cure may be brought about. The results of treatment in this class of cases have been most encouraging, as out of twenty-two cases operated on only one died directly from the operation, and in that case the patient's life was only very slightly shortened, since he was reduced to the last stage of exhaustion before the operation was performed. Of those recovering from the operation, with the exception of two that died a few months later, complete and permanent recovery ensued. These results contrast very markedly with the surgical treatment of cancer of the pancreas, where nearly half the cases operated on have died directly as the result of operation, and in those who have survived life has only been prolonged for a comparatively short time.

The Operative Treatment of Cirrhosis of the Liver.—FRAZIER (*Annals of Surgery*, June, 1901) reports the case of a man with marked cirrhosis of the liver who had been repeatedly tapped and whose condition seemed very

hopeless. Operation being decided upon as a last resort, the peritoneal cavity was opened. The parietal peritoneum of the abdominal wall on either side of the incision was rubbed quite vigorously with a gauze pad, and the omentum, which was very much thickened and contracted, sutured to the parietal peritoneum and to the margins of the wound. The fluid contents of the abdominal cavity were evacuated and the incision closed without drainage. Convalescence was uninterrupted; the patient suffered no ill effects from the operation. The wound healed throughout *per primam*. The history subsequent to the operation—since which three months have elapsed—is briefly as follows: The patient has been tapped twice, once on the thirteenth day, 328 fluidounces having been withdrawn, and again on the thirty-sixth day, on which occasion only ninety-six ounces were withdrawn. From that time to the present writing there has been absolutely no reaccumulation of fluid. The patient has gained rapidly in strength; he is no longer bedridden, goes out daily, and receives no medication other than enough citrate of magnesia to insure a daily evacuation of the bowel. This operation purports to open another channel for the relief of the obstructed portal circulation. The author says another channel, because there already exists a more or less free collateral circulation between the systems of the portal vein and the inferior vena cava. Thus, the coronary anastomose, through the œsophageal plexus, with the azygos veins; the veins of the cæcum and colon with the internal mammary; the hypogastric with the hemorrhoidal; the veins of the hepatic ligament with those of Glisson's capsule; the veins of the round ligament with the epigastric. By inciting the formation of adhesions between the omentum and the abdominal wall and between the surfaces of the liver and spleen and that of the diaphragm, this operation furnishes an additional outlet for the blood of the obstructed portal system. The examination of specimens obtained at the autopsy table has proved beyond a doubt that the operation as conducted accomplishes this purpose. Thus, in the case operated upon by Lens, venous channels were easily demonstrable in the adhesions that had formed between omentum and peritoneum.

The chief indication for the operation is the presence of ascites due to obstruction of the veins of the portal system, when the obstruction itself is due to cirrhosis of the liver. The operation is not indicated in every case of hepatic cirrhosis with ascites; the operation is absolutely dependent for its success upon the retained function of the liver cells. In other words, the absence of functional activity is an absolute contraindication. At the time of this writing the records of but fourteen cases (including the author's) have appeared in literature. The number of operations, therefore, is so limited that no very definite conclusions can as yet be drawn. When we exclude those in which there was some error of technique, those in which there was an error of diagnosis, or those in which the operation was contraindicated, but eight cases remain. Of these none died (mortality, 0 per cent.); one was living and unimproved (12.5 per cent.); one living and improved (12.5 per cent.), and six were living and free from ascites at periods of three, four, six, twenty-four, twenty-four, and twenty-six months respectively (75 per cent.).

This operation is indicated in those cases (1) in which the liver is cirrhotic; (2) those in which there is reason to believe the liver cells are not devoid of function; (3) those in which internal medication (particularly iodide of

potassium) and paracentesis fail to afford relief, or, in other words, in utterly hopeless cases, and (4) those in which there is no reasonable contraindication —the operation has a future. The cases are so hopeless, the technique so simple, the dangers so trivial, and the outlook so promising, that the prospects of this mode of treatment becoming an established one seem bright. That surgeons now have at their command a method of affording some relief in cases of intractable ascites, is evident.

The Application and Merits of Short-circuiting in Certain Diseases of the Intestines.—MAYLARD (*The Medical Chronicle*, April, 1901) states that the term "short-circuiting" is to be understood as the formation of a passage between one segment of the bowel above with another segment below, and thus the intervening portion of intestine is partially thrown out of action. There are two primary uses to which this method may be put—the one where it is desirable to divert the course of the intestinal contents past some obstructing influence, whether within or without the bowel; and the other where such diversion of fecal matter will admit of the closing of an artificial anus or fecal fistula. The author reports two cases of intestinal obstruction, both tubercular in origin, which made uninterrupted recoveries after this operation. This operation is indicated in cases of extensive malignant disease of the colon, and should prove a very valuable method of treatment. There are two methods by which short-circuiting may be executed —one by the lateral approximation and union of two loops of intestine and the establishment of a fistula bimucosa (entero-enterostomy, entero-colostomy, colo-colostomy), and the other by the division of a free segment of the gut, the closure of the distal end, and the lateral implantation of the proximal end into another free segment. The former method incompletely cuts off the passage of the intestinal contents through the partially occluded segment of gut, while the latter completely does so. Both methods have their own peculiar advantages, which are not immaterial in considering the particular method best suited for each individual case. Stitching should alone be employed in either method. In implanting the free end of the ileum into the colon it is as well to invaginate it for about half an inch. This is easily done by passing a traction stitch through the patent end of the ileum, and, after transfixing the colon at a couple of inches or so with the needle carrying the traction thread, pulling on the latter. While thus secured it is carefully stitched all around with a double circle of Lemberts. The traction thread is last withdrawn. The point requiring most attention is at the attachment of the mesentery to the invaginated end of the ileum.

In producing a fistula bimucosa the two portions of the gut involved in the union should be laterally approximated in such a way that the natural downward current of the feces is maintained. From two to three inches of the applied surfaces of the two segments of gut should be stitched first, this line of union forming the posterior boundary of the opening. Then the bowel wall of each segment is incised and the edges of the projecting mucous membrane of each orifice carefully united together, after which the posterior layer of stitches is continued on around the front until an external circle of Lemberts has been completed.

PEDIATRICS.

UNDER THE CHARGE OF

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Diphtheria Bacilli in Noma.—JOSEPH WALSH (*Proceedings of the Philadelphia Pathological Society*, June, 1901, p. 179) reports that he has found diphtheria bacilli in all of eight cases of noma examined bacteriologically. Only one of these cases gave a pure culture of the diphtheria bacillus, and this occurred in a case with associated pharyngeal diphtheria, the child subsequently recovering. Another developed noma during measles, having suffered from diphtheria four months previously, and at the time of development of measles being isolated with another child who also presented clinical symptoms of diphtheria, but with negative cultures as regards this organism. Three of the other cases began just after measles. Four of the eight cases began with an ulcerative stomatitis. Fifteen other cases of ulcerative stomatitis were examined in hope of finding diphtheria bacilli, but with negative results.

He concludes that since noma is a species of moist gangrene, requiring probably, from analogy, two different organisms—one a saprophyte, to produce the putrefaction; another a parasite, to produce the primary necrosis—it is possible that in the cases in which diphtheria bacilli are found they may be the primary causative agent; and, secondly, that when other pathogenic micro-organisms capable of producing necroses are found it is possible that they may be the primary excitants.

Confirmatory Evidence in Favor of the Fourth Disease of Dukes.—FREDERICK T. SIMPSON (*Archives of Pediatrics*, September, 1901, p. 693) adds some confirmatory evidence as to the existence of a fourth disease resembling in some features both rubella and scarlatina, a question which was raised about a year ago by CLEMENT DUKES (see abstract of Dukes' paper in this department of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, February, 1901, p. 233). An epidemic of twenty-seven cases of eruptive disease was observed by the author at the American School for the Deaf, in Hartford, in the spring of the present year. The symptoms generally resembled those of rubella, the rash in most of the cases being of the morbillous type. A slight branny desquamation followed the rash in the majority of the cases, but in eight desquamation occurred in strips or lamellæ—in three copiously and universally—in the others confined to the hands and feet. In one case the epidermis of the entire heel was separated like a cast. The cases to desquamate most copiously were of the scarlatinal type, except one of the morbilliform type. The throat was slightly injected in most of the cases, but in only three or four was there a subjective feeling o

soreness so far as could be ascertained. Enlargement of the post-cervical lymph nodes was a marked feature in every case. If looked for, the axillary and inguinal nodes were also found enlarged. True strawberry tongue was never seen, and vomiting occurred in only three cases. Six cases developed a second rash at from ten to fifteen days after the appearance of the first rash. One of these looked like a heat rash, but the others were coarse, discrete, and papular.

The points which justified the diagnosis of German measles as from scarlatina were: 1, absence of albumin in the urine in all cases; 2, known period of incubation of two weeks or more in five cases; 3, previous occurrence of scarlet fever in five cases, or 18 per cent.; 4, profuse development of rash in all cases, with slight disturbance of temperature and pulse; 5, absence of vomiting, strawberry tongue, angina, and of complications; 6, morbilliform type in two-thirds of the cases.

In the epidemic of nineteen cases at Rugby, described by DUKES (*Lancet*, July 14, 1900, p. 89), over 40 per cent. of the patients had already suffered from rubella, which, therefore, proved that the disease in question was not German measles. In the differentiation from scarlet fever, however, as was editorially pointed out by the *Lancet*, two important points were lacking: First, none of the patients had had scarlet fever, and, second, the period of incubation could not be established. In Simpson's cases, however, both these points were determined, so that it may be regarded as demonstrated that some cases of the affection hitherto known as German measles may desquamate like scarlet fever.

Apart from this interesting observation as to desquamation, of which one of the editors of this department can offer personal confirmation, Dr. Simpson advances evidence in support of Dukes' theory. He believes that in the epidemic he here reports two diseases were present instead of one, and that these two were rubella and the "Fourth Disease."

His evidence for this is as follows:

First, a number of patients, at least five, had a second rash. This occurred between two and three weeks after the first, lasted several days, was accompanied by a slight rise of temperature, but by no feeling of malaise. The rash was always coarse and discrete. It is difficult to account for these second rashes, except by assuming another disease, since relapses in German measles do not occur.

Second, four of the patients were stated to have had rubella previously. This has not the same probability as the statement that five of the pupils had had scarlet fever, for it will be recalled that quite a proportion of deaf mutes become so in consequence of scarlet fever. Nevertheless, the statement cannot be wholly discredited.

Third, Dukes has made the statement that a form of pink-eye may be the only symptom of rubella; that it may produce the ordinary form in another patient, and that it is protective against a second attack of rubella. An epidemic of pink-eye, affecting ten pupils, had occurred in the school shortly before the first case of the exanthematous epidemic. Three of these pupils had been subsequently affected.

Fourth, the fact of lamellar desquamation in a large proportion of cases of a disease which was certainly not scarlatina, does more than anything else

to make credible the existence of another eruptive disease. This form of desquamation is the chief diagnostic distinction between the fourth disease and rubella. If it belonged to rubella it would certainly have been noted long before Dukes' observations.

To restate the case: here is a series of cases of an eruptive fever having an incubation of at least two weeks, eight of which peeled in strips, five of which previously had had scarlet fever, and four, and possibly seven, had had German measles.

It is quite possible, therefore, that some of the mild epidemics of so-called scarlet fever occurring in the spring and summer are in reality the harmless affections which now pass under the name of German measles.

The Treatment of Intussusception in Children.—An interesting discussion on this subject before the British Medical Association, at its recent meeting, was opened by BERNARD PITTS (*British Medical Journal*, September 7, 1901, p. 574). The conclusions of his paper were formulated as follows:

1. Try inflation only when the case is seen within a few hours of onset, and is not of a very acute character. In the great majority of hospital cases it is better to open the abdomen at once.

2. Inflation may be tried in certain other cases for the purpose of reducing the main portion of the intussusception and enabling the incision to be made directly over the cæcum.

3. When reduction is found impossible in chronic cases a resection may be generally done through an incision in the ensheathing bowel.

4. In acute cases, and especially if gangrene is present or the condition of the bowel requires its removal, a wide resection should be undertaken as rapidly as possible, and the ends brought outside the abdomen; continuity should be restored at a subsequent operation.

5. In exceptional cases of enteric intussusception resection and immediate restoration of continuity gives the only chance.

He called attention to the fact that he had noticed after abdominal section in young infants, especially when there had been much manipulation of the intestines, that death is often preceded by high temperature and delirium, and takes place within twenty-four hours after the operation. Nothing is found post-mortem to explain the temperature or the death.

The general consensus of opinion of other hospital surgeons who discussed Mr. Pitts' paper was against the employment of inflation or injection, not even excepting the cases in which early recognition of the condition had been possible.

The Pathogenesis of Rhachitis.—RUDOLF FISCHL (*Archiv für Kinderheilkunde*, 1901, Hefte 5 u. 6) reviews the various theories that have been advanced to explain the etiology of this disease, but regretfully concludes that careful inquiry into the pathogenesis of rhachitis has failed to throw light upon the subject. On the contrary, he states, theories that hitherto seemed well-founded upon clinical observation and statistics have been found fallacious. He considers that Stölzner is right in holding that the future theory of rhachitis must be founded upon cellular pathology and biology.

THERAPEUTICS.

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Medication by Latent Arsenic.—DR. ARMAND GAUTIER under this title gives a résumé of his own and other researches on the therapeutic value of the cacodylates. He says that there are a large number of observations now on hand which justify for this form of medicament a settled place in therapy. They have been employed with benefit in a vast variety of diseases; grave anæmia, cachexia of various origins, tuberculosis in its numerous forms, pulmonary, osseous, and visceral, scrofulosis, herpetism, asthma, cutaneous diseases of many types, malaria, disturbances of menstruation, obesity, chorea, paralysis agitans, neurasthenia, myxœdema, scleroderma, exophthalmic goitre, syphilis, and sarcoma—truly a formidable array. In the matter of administration it is best given by the hypodermic method. Patients can bear five-sixths to one and two-thirds grains a day and continue such doses for at least two or three years without symptoms of arsenical poisoning or the occurrence of anatomical lesions in the viscera. The drug may also be administered by mouth, but it seems to be less well borne. This is interpreted as due to processes of oxidation and transformation of the cacodylates into more active forms of arsenic. He concluded his paper by giving the histories of a number of patients suffering from tuberculosis. These were much benefited, and the success of his treatment lead him to conclude that the cacodylates are capable of curing many patients with pulmonary and visceral tuberculosis, even when in seemingly advanced stages. —*Bulletin de l'Académie de Médecine*, 1901, vol. lxv., p. 20.

Action of Arsenic on Healthy Tissues of the Skin.—DR. LESLIE ROBERTS treats this subject from a broad biological point of view. He says that arsenic is universal in its distribution, being like nitrates and nitrites, a soil ingredient, and capable of entering into the tissues of plants and animals. It is closely associated with iron, nickel, and cobalt. In the human body arsenic combines with albumin, and excites the metabolism of cells to greater activity. Albumin and fibrin, according to the observations of Binz and Schultz, are capable of reducing arsenic acid to arsenous acid; blood exerts a powerful reducing action on arsenic acid, but has no action on arsenous acid. Fat exercises no action on the oxidation of this metal. The brain, liver, pancreas, and kidney all exercise a great activity. This arsenical oxidation is held by the author to be in all probability a normal function of

the skin, and he draws an analogy between the nutritive value of this metal for the skin and the thyroid for other types of metabolism. These metabolic activities are brought about by the formation in the tissues of nascent oxygen. He further draws other analogies, saying that perhaps Gautier's inference, that there exist in the body certain functional elements, of which very few have as yet been discovered, but which are essential to the performance of certain functions, is correct. Thus manganese is said to be essential to the oxidizing ferment; iodine to the thyroid; phosphorus to the nucleinic bodies; fluorine to the bone cells. Of the functional elements, some appear to play the rôle of "principal actors," while others may be termed substitutional elements or "understudies." Thus arsenic may, to some extent, take the place of phosphorus in relation to the nucleins; selenium is thought to be able to act as sulphur; copper, zinc, and manganese may replace iron; while phosphorus, arsenic, vanadium, or bismuth may play the rôle of nitrogen. Beyond these as yet theoretical assumptions, it is certain that arsenic is not merely an irritant poison, but its action is determined by the tissues themselves; that its effects are essentially of a nutritive order brought about by the agency of active oxygen; that these effects are of benefit to the organism when the oxidation is slow and detrimental when rapid; that the more highly organized the cell the more unstable its protoplasm, and the more rapid its metabolic processes, the more readily does it feel and manifest the action of arsenic.—*British Medical Journal*, 1901, No. 2126, p. 861.

Action of Arsenic on the Skin.—Dr. H. G. Brooke concludes from a series of microscopical studies that the main action of arsenic is obviously a stimulation of the growth and functions of the epithelium. The interpapillary pegs are very irregularly enlarged and prolonged downward, the prickle cell layer increased, the granular layer always and often very considerably thickened, the stratum lucidum is abnormally well marked, and the horny layer altered in character and unusually pronounced. As the poisonous action of the arsenic continues, either by the further absorption or the process of elimination of the arsenic through the skin, the hyperidrosis ceases and the horny layer becomes dry and lamellar, and scales off rapidly. Usually this is preliminary to restitution of tissue, but in pronounced cases the stimulation may have been so great that the cells are unable to recover, and atrophy of a more or less complete character sets in. The papillæ flatten, the cells lose their position and special characters until the whole epithelial layer is represented by three or four rows of degenerate cells, with a last trace of a stratum lucidum and a covering of very thin flat paper-like lamellæ. The sebaceous glands are not affected except by general atrophy, their fatty contents apparently rendering them immune to the action of the drug.—*British Medical Journal*, 1901, No. 2126, p. 860.

Quinine as a Prophylactic in Malaria.—Sir William MacGregor contributes an affirmative note to the discussion as to the value of quinine as a prophylactic. He thinks it highly probable that at least as much can be done by the use of quinine as by the employment of mosquito netting. The greater number of government officers take quinine regularly; but, he adds, so long as the taking of quinine is not compulsory there will always

be a residuum of men who, either because they cannot tolerate quinine or from some other reason, will not use it as a preventive of fever. The more common method in vogue in Lagos is to take daily doses of from two and one-half to five grains, but several take a large dose weekly and others irregularly. As a general prophylactic for the native he gives some interesting figures, showing that it would take seventy tons of quinine a year to efficiently protect the entire population of three millions at a grain a day regular dosing. This is an economic impossibility, and their efforts are being concentrated on the foreign population.—*British Medical Journal*, 1901, No. 2124, p. 680.

Prophylaxis in Malaria.—Dr. R. Blanchard, in an interesting and complete paper on the mosquitoes of Paris, shows that that city is more than well supplied with various species of Anopheles, and he formulates the following general rules for the prophylaxis of malaria, which follow closely those laid down by Laveran in 1900: 1. In quarters where Anopheles are found it is indispensable to make use of mosquito netting during the sleeping hours. Such mosquito netting arrangements should be so constructed as to permit of plenty of air and yet not admit the insects. 2. To protect the body from the bites of mosquitoes the author has found that the most efficient lotion is one from quassia—prepared by simple maceration in water. The various pomades and tinctures advised have been found less efficient. 3. To drive mosquitoes from the rooms he recommends the use of formaldehyde vapor, freshly generated from a lamp. This should be done about an hour before retiring. For those who are particularly susceptible to this vapor, pyretheum pastilles may be burned. 4. He further advises the use of wire screens. 5. To treat the actual sting, tincture of iodine is an efficient remedy. 6. The breeding places should be carefully drained.—*Bulletin de l'Académie de Médecine*, 1901, vol. lxv., p. 223.

Treatment of Malarial Fever by Iodine and Potassium Iodide.—Dr. A. Regnault, while recognizing the great value of quinine in the treatment of malaria, says that it is now becoming a matter of general recognition that the quinine-series of drugs is of service only during certain developmental periods of the disease. It is only while the parasite is undergoing its definite evolutionary history that these agents are poisonous enough to kill, and it is the practice at the present time, in France, at least, to administer quinine from six to eight hours preceding the onset of the fever, in order to cut short the stage of the division of the parasite. He also points out, by analogy, with certain septicæmic and pyæmic organisms, that the chill of malarial fever is to be interpreted as a chemical reaction to a toxin; and certain nervous manifestations of the disease, notably malarial polyneuritis, seem to corroborate this view. It is held that the toxins are developed with great rapidity just at the time of the division of the parasites, and the fever is therefore but a sign of the reaction to these protozoal poisons. It is with the idea of bringing about a chemical destruction of such poisonous bodies that the author has suggested the use of iodine and potassium iodide, iodine being a body which has a special affinity for bodies of the alkaloid class, and presumably for the supposed analogies of these

toxins. The results obtained are reported as being very striking, in that not only were the attacks aborted, but the action on the fever itself was striking and immediate, chills, vomiting, and malaise disappearing rapidly. The remedies were employed in the following strength: Tincture of iodine and potassium iodide, of each, 1; distilled water, 25. A teaspoonful in a little water at the beginning of an attack; a second teaspoonful fifteen or twenty minutes later, if required. In those patients in whom vomiting is a prominent symptom fifteen minims of ether may be added to the first dose. The use of Lugol's solution in chronic malaria is old, but this remedy has rarely been used in the attacks. It should be borne in mind that there is a chemical antagonism between iodine and quinine, and that when both are used enough time should elapse to permit of the elimination of either.—*Revue de Médecine*, 1901, vol. xxi., p. 804.

Antidotes for Cyanide Poisoning.—Drs. C. J. Martin and R. A. O'Brien, of Melbourne, commenting on the fact that cyanide poisoning had become not infrequent among the Australian miners since the introduction of the use of the cyanides in the extraction of gold, present a careful series of studies to determine, if possible, a practical course to pursue in the treatment of this condition. Hydrogen dioxide and the salts of cobalt have of late years been extolled, Kobert writing at great length of the value of the former antidote. Shoül first pointed out the desirability of forming insoluble cobalt cyanide. Ferrous hydrate has had much vogue in hereditary text-book writings. These investigators have studied these three agents in particular. Pure chemicals were employed, and rabbits were used in the animal experiments. In these animals 1 to 100,000 of potassium cyanide produced, within seven minutes, staggering gait, very rapid respirations, followed shortly by labored breathing; convulsions of varying intensity always occurred, and at times appeared within three minutes after the introduction of the drug. Exhaustion followed the spasms. Respiration then consisted of heaving movements of the chest and abdomen. The breathing became shallower and shallower, and the animals died. If the animal did not die within thirty minutes it usually survived. Hydrogen dioxide as recommended, subcutaneously in 3 per cent. solution and by the stomach in 2 per cent. solutions, was employed. When introduced subcutaneously it was immediately broken up, and its effects were *nil*—the animals all died; a similar result was obtained by its use by the stomach, even when the poison and its supposed antidote were introduced mixed. The authors say that hydrogen dioxide does oxidize cyanides, but such action is too slow to be of therapeutic value, and they detail a series of experiments to determine the velocity of this reaction ($2\,HCN + H_2O_2 = C_2O_2N_2H_2$). This interaction requires from twenty to seventy minutes, and any antidote for potassium cyanide must act instantaneously. Cobalt chloride was then studied in the same careful manner. Twenty-six experiments pointed to the fact that the cobalt chloride was capable of forming an insoluble cyanide, but that it must be given in excess; the acid of the gastric juice does not interfere with the reaction. Cobalt chloride itself, however, as pointed out by Stuart, is not free from poisonous action, producing severe gastro-enteric symptoms. This should be carefully guarded. Ferrous hydrate when added to a cyanide

salt forms a ferrocyanide almost instantaneously. Such ferrocyanides are but slightly poisonous, and its administration would seem to be even better than the cobalt salts, in view of their toxic action; but, unfortunately, the strongly acid character of the stomach contents greatly hinders this action, hence alkalies must be added to neutralize the free hydrochloric acid. The author's summary of results shows that there is no known physiological antidote for cyanide poisoning. Hydrogen dioxide is practically worthless, given in any form; cobalt salts are valuable, but are poisonous; ferrous salts administered with sufficient alkali are as efficacious as cobalt salts. At the body temperature the formation of ferrocyanides is instantaneous, but they possess two disadvantages: (1) They are kept in solution with difficulty, and (2) there is an absolute necessity of using enough alkali. This is best effected by the use of doses of magnesium oxide. In all mines where such processes are employed there should be kept solutions of ferrous sulphate, weak potash, and a small packet of magnesium oxide, together with a stomach-tube and a suitable receptacle for mixing, so that the remedies could be administered without delay, as time is the most important factor. They recommend an ounce of a 23 per cent. solution of ferrous sulphate; one ounce of a 5 per cent. solution of caustic potash; thirty grains of powdered magnesium oxide; a metal receptacle of one pint capacity; stomach tube. The first two solutions should be kept in air-tight tubes which can be broken into the receptacle, and powdered magnesia and half a pint of water added, shaken up, and administered. This amount of antidote will take care of seventy-five grains of potassium cyanide.—*Intercolonial Medical Journal of Australasia*, 1901, vol. vi., p. 245.

Malarial Parasites and Methylene Blue.—Dr. A. Iwanoff administers methylene blue in three daily doses of 5 grains each to patients in whose blood tertian parasites have been found. The first changes observed began at the end of the second day, and affected only the full-grown forms; the smaller, younger forms remain unchanged. The adult organism showed diminished movements and a breaking up of the protoplasm into lumpy-like bodies, which are rounded and lie on the blood cell apart from one another, or are connected with each other by minute bands of protoplasm and pigment granules. The protoplasm of these lumps is shrunken. The nucleus of the parasite is found generally near the peripheral portion of the blood-cell, and shows in its carmine-violet stained chromatin that it consists of numerous granules. Among the sporulation forms he found, beside normal sporozoites, others which were not fully formed. Some show their protoplasm very distinctly, while others appear to have no plasma at all. The pigment is gathered in patches, and these are irregularly distributed. The chief points he noted in the action of methylene blue on the tertian forms are destruction of the protoplasm and absence of alteration of the chromatin. In the parasite of æstivo-autumnal fever no changes were observed. In the crescent forms there was shrinking and granulation of the protoplasm. He noticed a more equal staining of the whole parasite than in the normal. The pigment granules appear coarser and are grouped in an irregular mass, which projects from the surface of the shrunken protoplasm. The destructive process of the protoplasm of malarial crescents goes on until there is noth-

ing of the parasite left except the pigment. Comparing the effects of methylene blue with those of quinine on the various forms of malarial parasites, it is said that the former affects the protoplasm, and the latter the pigment (chromatin). In the early stages the proportion of the pigment to the protoplasm is quite different to that in the adult, and thus the early forms are affected but slightly by methylene blue, while they are affected markedly by quinine, while the adult forms show the reverse.—*Deutsche med. Wochenschrift*, 1901, No. 18, p. 276.

GYNECOLOGY.

UNDER THE CHARGE OF

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Cancerous Infiltration of Fibromyoma.—GLOCKNER (*Centralblatt für Gynäkologie*, No. 37, 1901) reports an interesting case of adenocarcinoma of the body of the uterus complicated with intraligamentary fibroid. The entire mass was removed by abdominal section, the patient succumbing to septic peritonitis following perforation of the intestine at the site of a secondary cancerous nodule. Metastases were found at autopsy in the retroperitoneal glands, omentum, and intestines.

An examination of the specimen showed that the disease had penetrated the uterine wall at a point adjacent to the attachment of the fibroid, and had extended to the centre of the latter growth, a large cavity having been formed, which was lined with cancerous masses. The interior of the uterus was studded with nodules separated by islands of normal endometrium. The latter condition confirmed the reporter in his view that carcinoma of the uterine body often develops from a number of isolated nodules in the endometrium.

Origin of Uterine Fibroids.—BLAND SUTTON (*British Medical Journal*, April 6, 1901) reports a case in which a fibroid uterus the size of the fist was found to contain by actual count 120 fibromyomata, varying in size from a coriander seed to a cherrystone. He notes the fact that in every instance the nodule surrounded an artery, hence his inference that they originated in the muscular coat of the vessels. He formerly believed that they bore a close relation to the nerves.

Observations on Cancer of the Uterus.—BECKMANN (*Centralblatt für Gynäkologie*, No. 38, 1901) presents the results of his clinical studies in 437 cases of inoperable carcinoma of the uterus, which were under observation in the hospital in the course of two and a half years. These included forty-

three cases of recurrence after radical operations. The majority of the patients had borne several children. Dating from the appearance of the first symptoms of disease, the average duration of life was longer in women with recurrence following hysterectomy.

Primary Carcinoma of the Fallopian Tube.—BOURSIER and VENOT (*Revue de Gyn. et de Chir. Abdom.*, 1901, No. 2), in reporting a case of this rare condition, state that thirty-one have already been recorded. The ages of the patients varied from forty to sixty years. The menopause does not seem to be an important factor in the development of the disease. Salpingitis has a direct bearing upon it.

Retro-uterine Hæmatocele.—LEWINSOHN (Inaugural Diss.; abstract in *Centralblatt für Gynäkologie*, 1901, No. 38) gives the result of a study of eleven cases, in only four of which was the hemorrhage clearly due to tubal abortion. In one the blood came from a ruptured varicose vein on the surface of an ovarian cyst, but in the remaining six no cause could be assigned. Eight patients recovered without operation, the blood becoming entirely absorbed.

The writer states that Winckel never incises a retro-uterine hæmatocele, and has never lost a patient in consequence of this conservative treatment.

KOBER (*Centralblatt für Gynäkologie*, 1901, No. 39) reports two cases of cœliotomy for supposed ruptured ectopic gestation, in both of which extensive retro-uterine hæmatoceles were found. In both instances the tubes showed absolutely no evidence of gestation, and were clearly not the seat of the hemorrhage. In the first case it was probably due to excessive coitus; in the second to a violent muscular effort.

The writer does not approve of operation in the case of old hæmatoceles which tend to become absorbed.

Alcohol in the Treatment of Inflammation of the Pelvic Organs.—SCHMID (Inaugural Diss.; abstract in *Centralblatt für Gynäkologie*, 1901, No. 39) reports seventy cases of endometritis and diseased adnexa, which were treated as follows: The abdomen was covered with a compress saturated with 60 per cent. (later, 95 per cent.) alcohol, over which was placed rubber tissue, these being renewed thrice daily. Tampons saturated with 30 per cent. alcohol were inserted in the vagina every other day. The rest of the treatment consisted in lysol douches, rest in bed, massage and simple diet. At first there were some pain and elevation of temperature. 20 patients were cured; 11 were improved; 31 had a symptomatic cure, while the local condition was improved. 26 patients were treated with ichthyol tampons for the sake of comparison. Of these only 1 was cured; in 5 the symptoms were entirely relieved; in 9 there was improvement both in the symptoms and in the local condition; in 9 in the symptoms only.

Pseudoligamentary Ovarian Tumors.—SCHENK (*Centralblatt für Gynäkologie*, 1901, No. 13) calls attention to the fact that tumors of the ovary rarely develop between the folds of the broad ligaments, but more often behind them. To these he applies the term "pseudo-intraligamentary."

This condition is usually due to peri-oöphoritis, with resulting exudates which surround the growth. He reports a case which seemed to justify the inference that the inflammatory capsule is formed around the ovary before it undergoes cystic degeneration. It is sometimes exceedingly difficult to decide, even on careful examination of the specimen after removal, whether the neoplasm is intraligamentary or pseudo-intraligamentary.

Extension of Uterine Cancer through the Lymphatics.—PUPPEL (*Centralblatt für Gynäkologie*, 1901, No. 13), after careful studies of cancerous uteri, arrives at the conclusion that extirpation of the entire organ for cancer of the portio vaginalis is not justifiable. He found that the first lymphatics to be infected are those in the middle muscular layer. From this point extension of the disease takes place either to the vagina, or to the lymphatics of the broad ligaments adjacent to the original focus.

Metastases in the body of the uterus occur subsequently, the lymphatics of the inner muscular layer being first affected. Hence the deduction that high amputation, with resection of the broad ligaments, is the operation to be performed in cases of operable cancer of the portio, especially in elderly women.

Etiology of Hydrosalpinx.—POMPE VAN MEERDERVOORT (*Nederl. Tijdschr v. Verlosk en Gyn.; Centralblatt für Gynäkologie*, 1901, No. 13), reporting a case in which a large hydrosalpinx developed in a patient, aged fifty-four years, five weeks after curettement, explains its occurrence by the theory that the hyperæmia of the pelvic organs preceding the climacteric leads to an increase of the normal secretion of the tubal mucosa. In consequence of the subsequent senile involution of the tube its epithelial lining is lost, the opposite surfaces adhere, and the abdominal ostium is closed so that the contained fluid cannot escape and a hydrosalpinx results.

Errors in Diagnosis of Ectopic Gestation.—EDGAR (*Glasgow Medical Journal*, 1900, No. 6) reports a case of pelvic hæmatocele resulting from the rupture of hæmatoma of the ovary, two cases of pyosalpinx, and one of suppurating intraligamentary cystoma—all of which presented subjective and objective symptoms of extra-uterine gestation.

Salpingotomy.—GOVILLIOVD (*Lyon Méd.; Centralblatt für Gynäkologie*, 1901, No. 13) recognizes only two indications for this operation, viz.: (1) Cases in which the tube is thickened and its abdominal end is adherent and closed, the patient suffering so much pain that she is incapacitated from going about and attending to her work. (2) Chronic catarrhal salpingitis and hydrosalpinx. He does not approve of the conservative treatment of pyosalpinx except in those cases in which the pus is known to be absolutely sterile. Salpingotomy is unattended with risk, and recurrence is rare, while the possibility of future conception is a decided advantage.

Post-operative Peritonitis.—HINTZE (*Centralblatt für Gynäkologie*, 1901, No. 28) reports two successful operations for this condition. In a case of severe infection after Cæsarean section the abdomen was twice opened within twenty hours after the primary operation. The writer believes that the earlier interference is resorted to the better is the prognosis. The operator should confine himself to the removal of the masses of plastic lymph.

DERMATOLOGY.

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Ringworm from Canaries.—MACLEOD (*British Journal of Dermatology*, May, 1901), at a séance of the Dermatological Society of London, presented a photograph of a case of ringworm in a boy, aged nine years, of more than ordinary interest, owing to the unusual source of the infection. There was a well-defined plaque upon the back of the left wrist about two inches in diameter, reddish in color, uniformly elevated above the surrounding skin, with a slightly raised vesicular border. The patch, which had lasted about ten days, was peculiar in that it had not cleared up in the centre. The disease was traced to a pair of pet canaries which the patient was accustomed to feed upon his hand. The skin of the head and neck of both birds was bald, presenting here and there stumps of feathers, but was not inflamed. Microscopical examination of the stumps of feathers showed spores and fragments of mycelium. The spores were irregular in shape and size, and seemed to be less resistant than those of ordinary ringworm, breaking up readily in liquor potassæ, so that it was difficult to obtain satisfactory specimens. Attempts to cultivate the fungus were unsuccessful.

Disseminated Gangrene of the Skin in Children.—VEILLON and HALLÉ (*Annales de Dermatologie et de Syphiligraphie*, 1901, No. 5) report a case of multiple gangrene of the skin occurring in a child, aged eighteen months, following an attack of measles. The measles presented nothing peculiar, the attack being of ordinary severity; but four days after the appearance of the eruption lesions filled with a bloody fluid appeared upon the back of the neck, which were soon transformed into variously-sized, round and oval ulcers involving the entire thickness of the skin. These gangrenous ulcers rapidly extended, in some instances destroying large areas of skin. Besides these ulcers impetiginous, ecthymatous, and phlegmonous lesions occurred upon the forehead, scalp, and hand. Marked elevation of temperature and dyspnœa accompanied the disease, which terminated fatally at the end of two weeks. Bacteriological examination of the pus from a number of the lesions showed the presence of the staphylococcus aureus and an anaërobic bacillus previously described by one of the authors as the bacillus ramosus; microscopical examination of sections of the gangrenous tissue showed the same bacillus. The authors conclude that their case shows that disseminated gangrene of the skin in children is not an isolated affec-

tion, but that, clinically and pathogenically, it is a disease which should be placed in the class of true gangrenes, which bacteriology shows are due to the growth in the tissues of strictly anaërobic micro-organisms.

Multiple Ulcers of the Hands Due to Occupation.—HALL (*British Journal of Dermatology* June, 1901) reports the following case: A man, aged sixty-two years, suffered from an eruption of the hands of ten years' duration, characterized by hyperæmia, roughness, and branny desquamation; cracks and fissures also occurred about the knuckles and on the backs of the hands. The chief feature, however, was the presence of isolated ulcers on various parts of the fingers, which appeared from time to time, healing up after some weeks' duration or enlarging without any attempt at healing. These ulcers varied in size from a split pea to a sixpence, and were painful. The patient was employed in polishing electro-plate by means of finely-powdered lime mixed with a small quantity of olive oil, which, getting into the fissures in the skin, produced ulceration. When measures were taken to prevent this the ulcers speedily healed.

BROCQ and LAUBRY (*Annales de Dermatologie et de Syphiligraphie*, 1901, No. 4) describe a form of ulceration occurring upon the hands of those employed in dyeing skins, vulgarly called "*le pigeonneau.*" The affection is characterized by the presence of variously sized, round or oval, punched-out, quite deep ulcers situated upon the ends of the fingers and the dorsal surface of the left thumb. The pain accompanying the affection is usually quite severe, often causing the patient to cry out. The ulcerations take their origin in slight excoriations or fissures arising from any cause, and continue so long as the patient continues at his work, slowly extending in circumference and depth. Withdrawal from work is followed by healing of the ulcerations. The authors attribute the malady to the action of the irritating and mildly caustic substances contained in the various fluids employed in preparing the skins for dyeing.

Phlebitis Nodularis Necrotisans.—PHILIPPSON (*Archiv für Dermatologie und Syphilis*, Band lv., Heft 2) under the above title reports a case which exhibited lesions corresponding clinically with the so-called tuberculides of Darier. The patient, a girl, aged eighteen years, two years before coming under Philippson's observation, had had an eruption of painful pustules upon the legs, which after a time became ulcers and healed very slowly. When first seen there were a number of rounded ulcers surrounded by a marked halo, irregularly distributed over the legs, the bottoms covered with necrotic tissue. These began as inflammatory spots, which after a week or two became necrotic in the centre, forming ulcers which slowly spread peripherally. A few firm, bluish nodules were also observed on the heel and outer side of the foot. Microscopical examination of excised lesions showed that the process was an inflammatory one arising in the bloodvessels, the most marked changes being in the branches of the venous network which lies in the fat-tissue beneath the cutis. The author concludes from the study of this case that the tuberculides begin as a phlebitis.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Influence of Ozone on Bacteria.—In view of the inconsistent results arrived at by a number of experimenters on the bactericidal influence of ozone, DR. ARTHUR RANSOME and MR. A. G. R FOULERTON (*Public Health*, July, 1901, p. 684) undertook to investigate the subject anew, in the hope of coming to a definite conclusion with especial reference to the availability of this agent as a room disinfectant. The experiments were planned to ascertain whether ozone applied in large quantities, mixed with atmospheric air or pure oxygen, has a true bactericidal influence, and included tests of its action on the vitality of certain pathogenic and saprophytic bacteria and on the virulence of one pathogenic species (B. tuberculosis). The organisms employed in the several experiments included the following : B. tuberculosis, B. mallei, B. diphtheriæ, B. anthracis (sporing), B. typhosus, micr. melitensis, micr. candicans, B. coli communis, B. pyocyaneus, B. pneumoniæ (Friedländer), B. prodigiosus, staph. pyogenes aureus, str. pyogenes, saccharomyces albicans, sarcina ventriculi, and an anaërobic sporing butyric-acid-forming bacillus. The results of the experiments demonstrate that dry ozone has no appreciable action on the vitality of these organisms; that prolonged exposure does not diminish the pathogenic virulence of B. tuberculosis (in sputum), B. mallei, or B. anthracis; that ozone passed through a fluid medium containing bacteria has bactericidal power; "that any purifying action which ozone may have in the economy of nature is due to the direct chemical oxidation of putrescible matter, and that it does not in any way hinder the action of bacteria, which latter are, indeed, in their own way, working toward the same end as the ozone itself in resolving dead organic matter to simple non-putrescible substances."

Frequency of Trichinosis in the United States.—It is commonly stated that 1 to 2 per cent. of dissecting-room subjects contain trichinæ, but most of the observations upon which this is based fail to state that systematic microscopical examinations of all cadavers were made. Believing that trichinosis occurs more commonly than is generally thought to be the case, DR. HERBERT U. WILLIAMS (*Journal of Medical Research*, July, 1901, p. 64) made an extended investigation, in the course of which 505 cadavers, not selected as being more likely to be infested with trichinæ than any others, but taken at random, were examined. The proportion of cases which yielded positive results was much larger than was anticipated, being

no less than 5.34 per cent. In none of the cases was the infection very recent, as shown by encapsulation and calcification, and not one of the subjects died of trichinosis. The infections were of varying degrees of severity: sometimes very extensive and sometimes very slight. The birthplaces of the subjects included the most important countries of North America and Europe, but the number of cases was not enough to admit of accurate conclusions as to the influence of nationality upon the frequency of the disease. It is concluded that many cases of old trichinosis escape detection at autopsies, and that a large part of the published statistics are probably based on the naked-eye diagnosis, which will detect only the very severe infections.

Influence of Formaldehyde upon the Metabolism of Children.—Formaldehyde has come into more or less extensive use as a milk preservative, though it does not lend itself for the purposes of a general food preservative on account of its hardening influence upon certain tissues. As is the case with boric acid and borax, there is a decided conflict of testimony as to its effect upon the consumer; and since, in the absence of direct observations upon man, no conclusions can be drawn from experiments with young animals concerning the possible effects of small doses upon him, TUNNICLIFFE and ROSENHEIM (*Journal of Hygiene*, July, 1901, p. 321) concluded that the only way to gain information on this point was to make a series of metabolic experiments similar to those made in their investigations concerning the influence of boric acid and borax. These led to the following general conclusions: In doses up to 1 : 5000 in milk or 1 : 9000 in the total food and drink, formaldehyde exerts in healthy children no appreciable effects on the nitrogen or phosphorus metabolism or fat assimilation, but in larger doses or long continued it might tend to diminish phosphorus and fat assimilation, this effect being referable to an influence upon pancreatic digestion. In the above doses in healthy children it produces a retention of water in the body. In a delicate child in the above maximum doses it has a chemically measurable deleterious effect upon the nitrogen, phosphorus, and fat assimilation, again referable to an action upon the pancreatic digestion, combined with a slight intestinal irritant action, and increases the volume of urine and weight of feces. The excretion of lecithin in the feces is diminished, this effect being probably due to a stimulating action on the lecithin-splitting ferment of the pancreas. It exerts no appreciable intestinal antiseptic action, and has no influence upon the general health or well-being of children.

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